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THE BLALOCK OPERATION FOR CONGENITAL PULMONIC STENOSIS

Report of Two Successful Cases

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BLALOCK and Taussig, in 1945,[1] reported a successful operation for malformations of the heart in which there is pulmonary stenosis or atresia. Their work represented an important milestone in surgery and opened an entirely new field of cardiovascular therapeutics. Previously, a "blue" baby with a deformed heart was doomed to invalidism and early death, although isolated instances have been reported of such patients living many years. As a result of the work of Blalock and Taussig, it is now possible to transform many of these unfortunate cardiac cripples from a miserable existence to a life of comparative well being.

By far the most frequently encountered type of congenital heart disease associated with constant cyanosis is the tetralogy of Fallot,[2] which consists of the following associated cardiac defects: pulmonic stenosis or atresia, dextroposition of the aorta, interventricular septal defect, and right ventricular hypertrophy. Due to the pulmonic stenosis or atresia there is a marked decrease in the amount of blood which flows from the right ventricle to the lungs for aeration. Because of the dextroposition of the aorta the aorta receives blood from both the right and the left ventricles. Due to the interventricular septal defect there is an admixture of venous blood from the right side of the heart with the aerated blood in the

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left ventricle. The right ventricle becomes hypertrophied through its efforts to force blood through the stenosed or atresic pulmonic outlet.

The cause of the constant cyanosis which is the outstanding symptom of these patients can be readily understood from a consideration of the above cardiac defects. The primary cause of the cyanosis appears to be a deficient circulation of blood to the lungs for aeration. Additional causes are the admixture of venous and arterial blood which occurs because of the dextroposition of the aorta and because of the interventricular septal defect. It has been estimated that when 30 per cent or more of the blood sent into the systemic circulation is venous blood there will be cyanosis.[3] That deficient circulation of blood to the lungs is the prime factor in the production of the cyanosis in these patients is conclusively shown by the fact that the Blalock operation, which increases the blood flow to the lungs, greatly relieves the cyanosis.

As a result of the poor circulation to the lungs there is a lowered oxygen content of the arterial blood. This leads to a compensatory polycythemia with marked increase in red blood cells and hemoglobin, so that it is not uncommon to find values for red blood cells and hemoglobin 50 per cent higher than normal, or even more. This polycythemia may be an additional factor in the production of cyanosis, for Lundsgaard and Van Slyke[4] have shown that in most, if not all, cases in which there was a pronounced polycythemia, secondary changes occur in the lungs of such a nature that all of the blood that passes through the lungs is no longer in effective contact with the oxygen in the alveoli. Hematocrit determinations in these cases show elevated figures indicative of an excess of the solid elements of the blood. Clubbing of the fingers and toes is another manifestation of the anoxia of the tissues present in this condition.

Disability in the tetralogy of Fallot depends upon the degree of oxygen unsaturation of the arterial blood. The milder patients become dyspneic only after extensive exercise. The severe patients are often so incapacitated that a few steps bring about acute respiratory distress. These children with deep cyanosis characteristically lie on their chests with their knees drawn up under them to make breathing easier. After walking a few steps they assume a squatting position until the dyspnea passes off.

Examination of the chest usually reveals a harsh systolic murmur heard best anteriorly over the third or fourth interspace.

X-ray examination of the chest is important and necessary in order to arrive at the correct diagnosis. In the tetralogy of Fallot, a plain chest film reveals a concavity of the heart shadow on the

left, in the region of the pulmonary artery, and no congestion in the lung fields. Careful fluoroscopic examination shows an absence of pulsations in the hilar regions. These chest findings should be present before operation is considered. They signify that the pulmonary artery is not pulsating forcibly and that the pressure in that vessel is low. In addition to this information, fluoroscopy in conjunction with a swallow of barium[5] will enable one to determine the position of the arch of the aorta by revealing the point where the aorta makes pressure upon the esophagus. The aortic arch will be found on the right side in an appreciable number of cases. Dysphagia[6] is sometimes a symptom of right aortic arch. The electrocardiogram in the majority of cases shows right axis deviation due to the increased work of the right side of the heart.

Prior to the work of Blalock, patients with severe tetralogy of Fallot were considered hopeless. An unsuccessful attempt was made by Doyen[7] in 1913 to aid a patient surgically by incision of a stenotic pulmonary valve with a tenotomy knife. In 1939, Levy and Blalock[8] successfully anastomosed the end of the left subclavian artery to the distal end of the divided left pulmonary artery in order to study the effects produced by the shunting of systemic blood to the left lung. In 1941, Eppinger, Burwell, and Gross[9] anastomosed the end of the subclavian artery to the side of the pulmonary artery in dogs in order to learn the effects of the ductus arteriosus which they thus created. In 1945, Blalock[1] used for the first time the end to side subclavian and pulmonary artery anastomosis in children with the tetralogy of Fallot.

The Blalock operation for congenital pulmonic stenosis[1] has for its purpose the increase of blood flow to the lungs. The pulmonic stenosis which is present in the tetralogy does not permit the lungs to receive sufficient blood for aeration. As a result, the left heart receives poorly oxygenated blood from the lungs for distribution through the systemic arteries to the tissues of the body. By surgical means the end of one of the systemic arteries arising from the aortic arch was anastomosed by Blalock to the side of either the right or left pulmonary artery, thus creating an artificial ductus arteriosus.

The approach is made through either the left or right side of the chest, depending upon which side the aortic arch is on. The present concept[10] is that of utilizing the subclavian artery which arises from the innominate, whenever possible, since a more suitable angulation of the vessel will be obtained than if the subclavian from the aorta were used. In cases of left aortic arch, as revealed by roentgenograms and fluoroscopy, the innominate artery will be found in the right side of the chest. Where an anomalous right

aortic arch is present, the approach should be made through the left chest, as the innominate artery will be on the left. Necessity occasionally demands the use of the common carotid artery for the anastomosis, because of an abnormal aortic arch, but its use should be avoided if possible because of the occurrence of cerebral damage[11] in some cases as a result of decreased circulation to the brain.

The following are detailed reports of 2 cases of pulmonic stenosis in which the Blalock operation was carried out. The surgical technic was worked out on dogs before performing the surgery on these children.

CASE 1. C. M. D., a colored girl of 4½ years, was said to have been cyanotic at birth even when not crying, but when crying became exceedingly blue. The child sat up at 6 months, talked at 18 months and walked at 2½ years of age. There had been no illnesses other than colds associated with a cough and deepening cyanosis. She could run only 20 steps and would then stop, gasp for breath, and later go in and lie down. She could walk very slowly almost a city block but became very dyspneic in doing so. She spent most of her time in bed and was quieter and more serious than the other 4 children in the family. Her favorite position in bed was the knee-chest position. Two months before admission to the hospital she had begun to lose weight and her appetite to diminish. From that time on she rarely left her bed.

The patient was admitted to the John Gaston Hospital on Aug. 19, 1946. Physical examination showed that she was slightly undernourished. She was very shy and serious and would not answer when spoken to. The respiratory rate, while sitting in bed, was more rapid than normal. The fingernails and toenails were blue and there was clubbing. There was marked cyanosis of the lips and buccal mucous membrane. Her admission weight was 29 lbs. 13½ ounces. The heart did not appear to be enlarged on physical examination. The blood pressure was 145/130. A harsh systolic murmur was heard over the entire precordium, but was especially prominent at the parasternal line in the fourth left interspace.

Her red blood count on admission was 7,190,000; the hemoglobin 23.5 Gm. On September 2, her red count was 7,170,000 and hemoglobin 27.5 Gm.

X-ray examination of the chest showed some cardiac enlargement with right ventricular hypertrophy. No fullness of the pulmonary conus was seen (fig. 2). Fluoroscopy with a swallow of barium showed that the aorta descended on the left side, and there were no visible pulsations in the lung fields.

The electrocardiogram revealed right axis deviation.

The diagnosis was made of tetralogy of Fallot with left aortic arch, and operation was decided upon.

Operation was performed on September 19, with the assistance of Dr. O. B. Stegall and Dr. Carruthers Love. Dr. Robert Hingson administered the anesthetic of cyclopropane and ether with a closed system. Little ether was used, and 90 per cent oxygen was given with 10 per cent cyclopropane.

The approach was made in the third left interspace to utilize the left subclavian artery arising from the left aortic arch. The incision extended from the left border of the sternum to the axillary line. The cartillages of the

third and fourth ribs were severed and the ribs were retracted with an automatic spreader. Ample exposure was afforded. A large number of small collateral vessels were seen about the hilum of the lung and the surface of the lung itself. The pulmonary artery, which was found to be in normal position, had no palpable pulsation. The left subclavian artery was isolated and it appeared to be of sufficient length and size for the purpose of anastomosis. The artery measured approximately 7 mm. in diameter. A bulldog clamp was applied to the subclavian artery at its point of origin and the subclavian

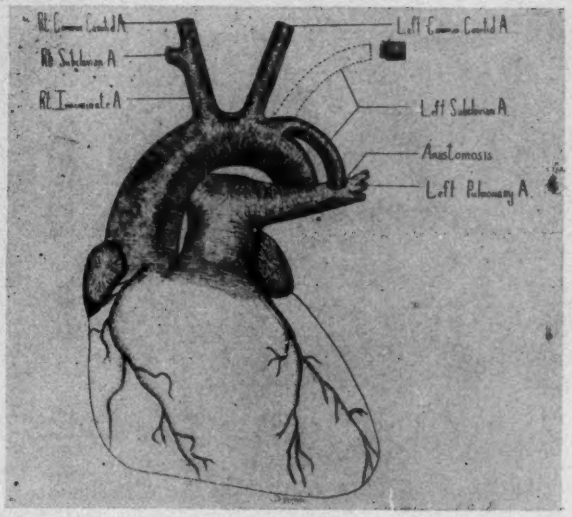

Fig. 1. (Case 1) Operative procedure. Aortic arch on the left. Anastomosis of the end of the left subclavian artery to the side of the left pulmonary artery.

was ligated and divided just proximal to its junction of branches. The adventitia was cleared from the open end of the artery and a circular patch of adventitia was removed from the pulmonary artery at the proposed site of anastomosis. Bulldog clamps were applied to the proximal and distal parts of the pulmonary artery and the pulmonary artery was incised. An anastomosis was performed between the end of the subclavian and the side of the pulmonary artery using an everting, continuous suture of 5-0 arterial silk with several interruptions.[1] The operation is shown diagrammatically in Figure 1. Construction of the anastomosis was very difficult because of inadequate space between clamps on the pulmonary artery and it was obvious that a special clamp was needed for the proximal part of the pulmonary artery. The pulmonary artery was clamped off for a total of 90 minutes. One persistent bleeding point on the anterior wall was controlled with mattress sutures. On the final removal of all clamps dilatation of the pulmonary artery was seen to take place and a thrill was palpable the entire length of the pulmonary artery. The chest wound was closed with silk and the ribs were fixed by an encircling suture of silk. At the completion of the operation the left pleural cavity was aspirated for blood and air by an indwelling catheter while the anesthetist reexpanded the lung. The catheter was then removed. The

patient's condition after operation was good and the cyanosis was somewhat less intense than prior to operation. She received 150 c.c. whole blood, 40 c.c. 10 per cent glucose and 300 c.c. plasma during the operation through a cannula in an ankle vein.

Postoperative Course. The postoperative course was moderately smooth. The child was kept in an oxygen tent for 4 days. The left chest was tapped on the third day and only 65 c.c. of bloody fluid aspirated. No further paracenteses were performed and the left lung reexpanded in a few days (fig. 2). Penicillin which had been begun 2 days prior to operation was dis-

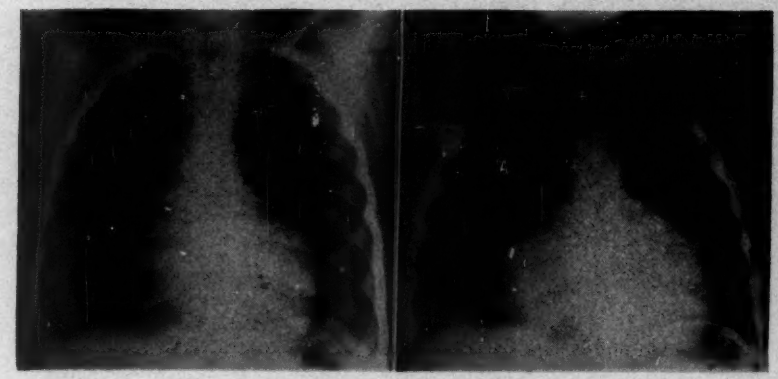

Fig. 2. (Case 1) Appearance before and after operation: A, before operation; B, after operation.

continued after 2 weeks. Her temperature was normal after the tenth postoperative day.

Although a satisfactory thrill was present in the pulmonary artery after release of the clamps, a new murmur did not develop in the left chest for several days. In the meantime the patient's cyanosis had begun to diminish perceptibly the night of operation and by the eighth postoperative day her lips, mucous membranes and nail beds were pink. On the tenth day a soft continuous murmur was heard for the first time over the left chest in the second interspace anteriorly and over the entire upper chest posteriorly. This murmur gradually became louder until the fifteenth day when it reached its maximum intensity.

Her red blood count on September 30, eleven days after operation, had decreased from 7,190,000 to 6,550,000 and the hemoglobin from 23.5 Gm. to 18.5 Gm. On October 4 her red blood count was 6,270,000 and her hemoglobin 18 Gm. A red count on October 13 shortly before discharge from the hospital was 6,000,000 with 16.5 Gm. of hemoglobin. The hematocrit on this date was 53.

She was permitted out of bed on the thirteenth postoperative day and she walked 35 feet without difficulty and without return of cyanosis. Exercises were increased until discharge from the hospital on the twenty-ninth postoperative day, at which time she could walk about as desired and play with the other children without any demonstrable handicap. The oxygen saturation of the arterial block at this time was 74 per cent.

The child was seen again on November 20. She had gained 4 pounds in weight since the operation and had developed an enormous appetite. The mother stated that the child played outside all day and was rapidly losing her shyness.

CASE 2. C. W., a 4-year-old white boy, was born Dec. 7, 1941. He was a full-term child, the birth weight being 8½ pounds. A few hours after birth it was noted that he was blue. The cyanosis then disappeared and the baby was considered normal. At 13 months of age he had a severe attack of dyspnea

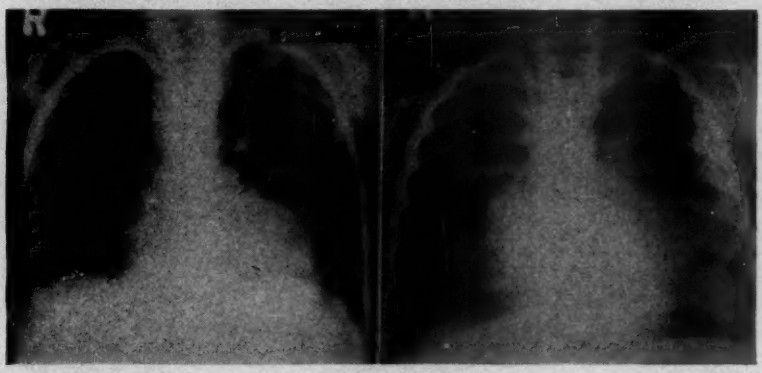

Fig. 3. (Case 2) Appearance before and after operation: A, before operation; B, after operation.

accompanied by intense cyanosis. He rolled his eyes upward and became unconscious. From this time on he remained cyanotic. Attacks of dyspnea and unconsciousness would appear following eating or moderate exercise, one bite of food sometimes bringing on an attack. There would be an average of 1 to 3 such attacks each week. On several occasions blood was noted in the stools. At 3 years of age the cyanosis began to increase and the child became more incapacitated. For the two months prior to admission to the hospital he was in even worse condition, having severe attacks of dyspnea and intense cyanosis several times daily.

Family History. The mother was a deaf mute. The mother's brother was also a deaf mute. There was no heart disease in the family. According to the father, the patient learned to converse with his mother in sign language at the age of 1 year, before he could talk.

The patient was admitted to the Baptist Hospital on the night of Oct. 4, 1946, in acute distress. He was gasping for breath and unconscious. His buccal mucous membrane and lips were blue-black in color. The fingernails and toenails were deeply cyanotic and clubbing was present. The skin over the entire body had a bluish hue and many small collateral veins were seen, especially on the face. The heart rate was too rapid to count. He was placed in an oxygen tent and in a short while began to breathe more easily.

A more detailed examination was performed the following day when his condition had improved. He was considerably undernourished, weighing 23½ pounds. His blood pressure was 104/78. The cardiac apex was in the fifth left intercostal space, just inside the nipple line. There was a harsh systolic

murmur heard best in the third left interspace at the sternal border. The pulmonic second sound was clearly heard. The heart did not appear to be enlarged. There were palpable pulsations in both femoral arteries.

X-ray examination and fluoroscopic study on October 7 showed the heart to be of normal size with a concavity to the left of the sternum in the region of the pulmonary artery. The superior vena cava was seen to the right of the sternum as a widened shadow (fig. 3). There was no visible pulsation of the pulmonary artery and no congestion in the lung fields. After the admin-

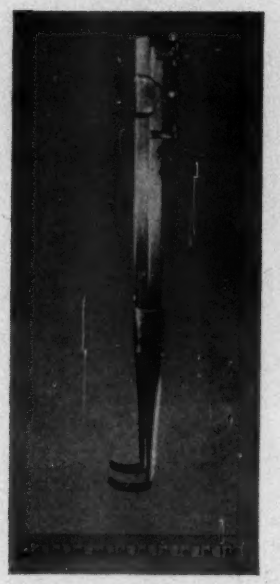

Fig. 4. Special clamp used to occlude the pulmonary artery during the anastomosis.

istration of barium the aortic arch was seen to be on the right side with indentation of the esophagus to the left.

The red blood count was 10,480,000; the hemoglobin was 25 Gm.; the hematocrit was 78.

The electrocardiogram showed a right axis deviation and changes suggestive of coronary insufficiency.

The diagnosis was made of tetralogy of Fallot with probable pulmonary atresia and progressive closure of the ductus arteriosus.

Operation was performed on October 17. Before anesthesia was begun an attempt was made to obtain blood from the femoral artery for the determination of arterial oxygen saturation. The puncture was unsuccessful and the patient became intensely cyanotic and upset so it was decided to forego the test and begin the anesthetic.

Dr. Robert Hingson administered cyclopropane with a high oxygen content through an endotracheal tube. Surgical assistance was given by Dr. O. B. Stegall and Dr. Carruthers Love.

In view of the presence of a right aortic arch in this case, it was decided to utilize the subclavian artery arising from the innominate and the approach was through the left chest. The incision was made in the third left intercostal space from the edge of the sternum to the axillary line. There was

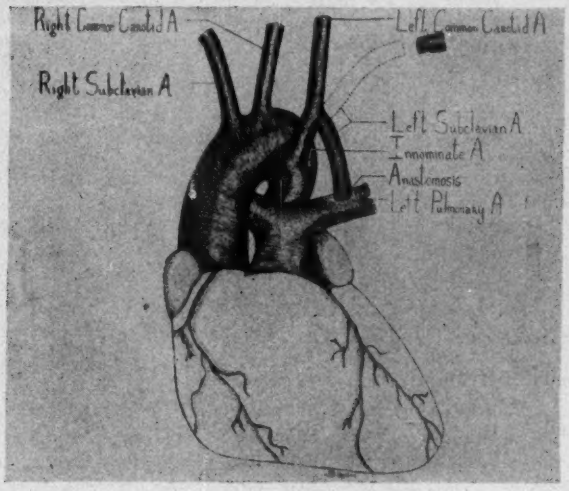

Fig. 5. (Case 2) Operative procedure. Aortic arch on the right. Anastomosis of the end of the left subclavian artery arising from the innominate artery, to the side of the left pulmonary artery.

no visible aorta in the left chest, substantiating the diagnosis of right aortic arch. The left pulmonary artery, which was in normal position, was easily compressed and had no palpable pulsation. Surprisingly, there were no unusual collateral veins surrounding the pulmonary artery. It was expected that they would be present because of the marked collateral vein formation on the body. The subclavian artery was identified as it branched from the innominate. It was temporarily compressed with umbilical tape while the anesthetist checked for the presence of the temporal pulse on that side to be certain that the left common carotid artery was not being used. The pulmonary artery was then dissected free and occluded for 3 minutes with a bulldog clamp to be sure that this was not the only pulmonary artery present. The subclavian artery was ample in size, measuring approximately 8 mm. in diameter. A bulldog arterial clamp was placed on the subclavian artery at its origin and the vessel ligated and divided just proximal to its branching in the upper chest. A special right angle clamp (fig. 4) was used to occlude the pulmonary artery proximally and small bulldog clamps were placed on the distal branches. A transverse incision was made in the left pulmonary artery, and an anastomosis was performed between the end of the subclavian artery and the side of the pulmonary artery using 5-0 arterial silk on a small, curved, atraumatic needle. A diagram of the completed operation is shown (fig. 5). The continuous everting suture was employed with several interruptions. After removal of the clamps the pulmonary artery was seen to dilate appreciably and there was a pronounced thrill present in the pulmonary artery both proximal and distal to the anastomosis and out in the lung itself.

The chest wound was closed with interrupted silk sutures. A catheter which had been left in the pleural cavity until the chest wall was closed was aspirated while the left lung was slowly reexpanded by the anesthetist. The catheter was then removed.

The patient's condition after operation was good. Due to Dr. Hingson's expert administration of approximately 90 per cent oxygen and only 10 per cent cyclopropane his color was better under anesthesia than before operation. He received 185 c.c. of normal saline, 165 c.c. of 5 per cent glucose, and 170 c.c. of plasma through an ankle vein during the operation. At one point when the operation was in progress his heart rate slowed to 80 beats per minute. Atropine was given intravenously and the heart action improved immediately.

Postoperative Course. The postoperative course was surprisingly smooth. The patient was placed in an oxygen tent on return to his room. On the evening of operation a loud, continuous murmur developed in the left second interspace anteriorly and it was heard over the entire posterior chest on both sides.

Penicillin was begun the day of operation and discontinued on the fifth postoperative day. The oxygen tent was discontinued on the second postoperative day as the child breathed as well out of the tent. His temperature became normal on the fifth day and there was no necessity for tapping his chest.

There was a rapid improvement in color each day, and, peculiarly, his toenails became pink before the fingernails. The buccal mucous membrane and lips were of approximately normal color by the fifth day.

The polycythemia decreased quite rapidly also. On October 19, two days after operation, his red blood count had decreased from 10,480,000 to 7,170,000 and the hemoglobin from 25 Gm. to 16.8 Gm. On the fifth day his red count was 6,130,000, the hemoglobin 15 Gm. and the hematocrit had decreased from 78 to 53.

A roentgenogram of his chest on October 21 showed a convexity in the region of the pulmonary artery, slight enlargement of the heart, and normal congestion in both lung fields (fig. 3).

In an effort to determine the possible body loss of the iron in the destroyed red cells serum bilirubin and stool specimens for blood were examined with negative findings.

Although he had been sitting up in bed from time to time since the second postoperative day he was not permitted out of bed until the tenth day. At that time he walked 15 feet and his legs became unsteady. However, his color remained unchanged and there was no dyspnea. He was permitted to walk further each day and his strength returned rapidly. He was discharged from the hospital on the sixteenth postoperative day. By then he could walk 100 feet without effort. His color had remained good and at no time was a return of cyanosis noted. The oxygen saturation of the arterial blood was 75 per cent.

were made at the time of the patient's discharge from the hospital. He reported that the right axis deviation persisted and the changes in the S-T positions were more characteristic of coronary insufficiency (fig. 6).

In view of the changes in the preoperative electrocardiogram, Dr. Jacob Alpèrin was asked to give an opinion on the postoperative tracings, which

The child was seen again on November 21, approximately one month after operation. He had gained 3½ pounds and had an excellent appetite. His color had improved even more with only a slight tinge of blue remaining in

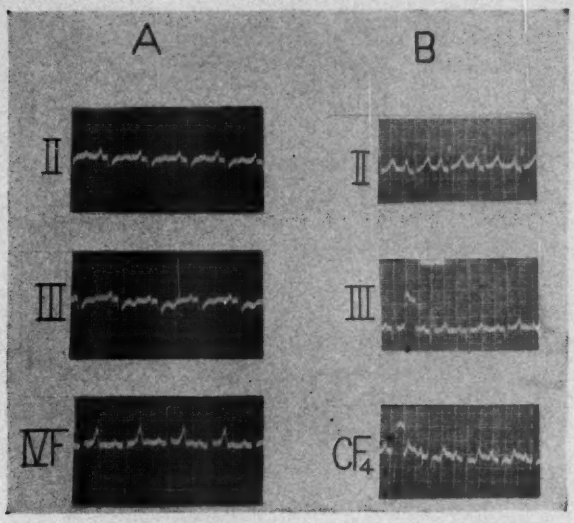

Fig. 6. (Case 2) Electrocardiograms showing coronary insufficiency: A, before operation; B, 12 days after operation.

his fingernails. The clubbing of the fingers and toes had greatly diminished. He played about the house without any return of cyanosis or dyspnea.

Discussion

Both patients have been greatly benefited by the operation. In both the cyanosis has disappeared with gradual recession of the clubbing of the fingers and toes. The polycythemia has decreased and their tolerance to exercise has increased rapidly. They have developed continuous murmurs in the left chest as evidence that the anastomoses are functioning.

It is interesting to speculate on several aspects of this operation, one being the fate of the iron in the destroyed red cells when the polycythemia disappears. Our second patient lost 10 Gm. of hemoglobin per 100 c.c. of blood and over 4,000,000 red cells from the circulating blood stream during the first 4 postoperative days. Nevertheless there was no increase in the serum bilirubin during the first postoperative week, nor was there iron lost in the stools. Granick[12] points out that tracer studies indicate large amounts of iron in the ferric state can be taken out of circulation by the liver within a few hours' time and some goes directly to the bone marrow.

Tissues which removed iron most actively from the blood stream are those which contain numerous phagocytes. The plasma protein iron is in equilibrium with the iron of the liver, spleen and bone marrow. Damaged red cells are destroyed by the phagocytes of these organs. The iron released from the hemoglobin breakdown is carefully conserved by the body to be re-used.

The response of our cases under anesthesia was gratifying. Dr. Hingson carried both patients with inhalation of only 10 per cent cyclopropane. A small amount of ether was given in the first case. The high percentage of oxygen afforded the patients more oxygen than they normally were able to obtain, with the result that their color improved under anesthesia. It might be added that the help of an expert anesthetist adds materially to the safe surgical conduct of this type of case.

The end to side anastomosis, the end of a branch from the aorta to the side of one of the pulmonary arteries, appears to be a satisfactory one, provided that the systemic artery is of sufficient length so that tension will not be present at the anastomotic site. There is the added advantage of the passage of aortic blood under greater pressure to the pulmonary artery of lesser pressure to assist in the continued patency of the anastomosis.

It should be mentioned that there is the danger of anastomosing the systemic artery to the pulmonary vein where the latter is in an anomalous position.[10] Due to the absence of palpable pulsation in the pulmonary artery in cases of stenosis one cannot rely on palpation for selection of the artery. An anastomosis between a systemic artery and pulmonary vein would result in a rapidly fatal pulmonary edema. Manometric study of the vessel in questionable cases would serve to differentiate the artery from the vein. Should there be only one pulmonary artery its occlusion for sufficient time to perform an anastomosis would also produce a fatal result. This can be avoided by applying a temporary clamp to the pulmonary artery for 3 or 4 minutes before severing the systemic artery.[10]

Neither heparin nor dicoumarol was used in the cases reported. We considered that the danger of postoperative bleeding outweighs the danger of thrombosis at the anastomotic site. It is perhaps preferable to rely on the greater pressure of the systemic artery for patency of the anastomosis.

Atropine[10] is a valuable adjunct for preoperative administration in these cases. In our second case a perceptible slowing of the heart occurred at one point during the operation and caused some concern. The intravenous administration of atropine produced prompt return of the normal rate. The drug's action, that of depressing

the parasympathetic nerve endings, may be achieved also by injection of the vagus nerve in the operative field.

It should be understood that there are several conditions associated with persistent cyanosis which would not be benefited by the operation for pulmonic stenosis. The first of these is "tetralogy of Fallot of the Eisenmenger type." The cyanosis, in this type of case, is probably due to changes in the alveolar wall or in the pulmonary vascular bed, which slow up the aeration of blood in its passage through the lungs. There is no inadequacy in the amount of blood transported to the lungs. Complete transposition of the great vessels may also be associated with cyanosis. In this malformation the aorta arises from the right ventricle and the pulmonary artery from the left ventricle. The blood from the right ventricle is forced out through the aorta and returns to the right side of the heart. Blood from the left ventricle is transported to the lungs through the pulmonary artery and returns to the left side of the heart. Hence the blood is well aerated in the lungs but reaches the systemic circulation only with difficulty. The cyanosis of aortic atresia is dependent upon the difficulty of forcing blood into the systemic circulation. Such blood that does reach the systemic circulation has been sent through the ductus arteriosus before reaching the lungs and therefore is not entirely oxygenated. It has been suggested that many cases diagnosed as interventricular septal defect are actually aortic or subaortic stenosis.

Summary

Two cases are reported in which the Blalock operation was done for the relief of pulmonic stenosis.

Both cases were done recently and the follow-up is a short one. However, the results have been promising. In both patients the cyanosis has disappeared and the polycythemia decreased to almost normal values. There has been an increased tolerance to exercise, and there is no visible evidence of disability in these two patients.

It is too soon to evaluate the future outcome of these operations for pulmonic stenosis. No one can predict whether or not subacute bacterial endarteritis will be a frequent complication. The fact remains that most patients with severe pulmonic stenosis die at an early age from anoxia or cerebral thrombosis. Since the operation is used primarily for the severe case it would appear that relief from the disability of pulmonic stenosis and the prolongation of life should be the major consideration.

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RETROPERITONEAL CHYLOUS CYST

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RETROPERITONEAL cysts which contain chyle are considered to be among the rarest tumors encountered in general surgery. Judd and Heimdal[1] reported eleven cases from the Mayo Clinic and a few authors have reported several cases each, but the majority have presented single case reports. Parsons[2] was able to collect about 500 cases from the literature; in some of them, however, he stated that the diagnosis was open to question. The great majority of cysts of this type occur within the mesentery, those situated in other locations retroperitoneally are exceedingly rare and only 18 such cases, according to Gerster[3] were found in the literature up to 1939. Carter, et al.,[4] reported an additional case.

The following instance of such a chylous cyst, situated retroperitoneally but not within the mesentery, is reported, therefore, because of its extreme rarity and other features of interest.

CASE REPORT

A woman, para II, aged 43 years, was seen in 1941 and in May, 1946, for routine examinations, which were normal. There had been no previous operations or illnesses of note. In August, 1946, she reported that for one week previously she had been aware of a mass in the right upper abdomen, noted particularly on first lying down. No associated symptoms were present and the physical examination was negative. A mass was not palpated at this time and she was asked to return in two weeks for further study. On Aug. 28 she was still aware of the mass; there were no other complaints.

The patient was a healthy, well nourished female. At this time, on first lying down, a mass was palpable in the right upper abdomen. It became less apparent on further palpation and was not demonstrated in the erect position. It was about the size of a grapefruit, smooth, movable and non-tender. Intravenous pyelograms and fluoroscopic examination of the colon and chest were normal. The hemoglobin estimation was 10.3 Gm. per 100 c.c. and the urine was normal. An omental or other rare type of tumor was suspected and exploration advised.

At operation, through a right rectus incision, a large multilocular retroperitoneal tumor presented in the right abdomen within the hepatic flexure of the colon and lateral to the superior mesenteric artery. It consisted of a larger intra-abdominal and a smaller portion situated deep in the retroperitoneal space between the retroperitoneal duodenum, the head of the pancreas and the inner aspect of the right kidney. It was lobulated, roughly dumbbell-shaped, ivory colored and traversed by small blood vessels. The attachments to the surrounding structures were by loose fibrous tissue which became noticeably more dense near the head of the pancreas. The overlying peri-

toneum was intimately attached to the cyst; no actual connections were demonstrated with any structures or organs. A separate tiny cyst of similar appearance, approximately .4 cm. in diameter, was present near its pancreatic portion. The cyst was easily enucleated by blunt and sharp dissection,

Fig. 1: Retroperitoneal chylous cyst partially emptied. Note multilocular appearance, thin walls with vascular network.

although it was ruptured in several places when the more intimately attached peritoneum was separated. The opening in the peritoneum was sutured and the abdomen closed without drainage. The convalescence was entirely uneventful and four months later the patient remained well.

Pathologic Examination

By J. D. Bush, M.D.

The cyst is multilocular, red-white (fig. 1), and measures approximately 15 cm. by 15 cm. by 5 cm. The cystic spaces are filled with milky fluid and apparently do not communicate with each other. The walls are paper-thin

and contain a coarse network of moderate and small sized blood vessels; the lining of each cavity is smooth and glistening and no villus projections are apparent.

Microscopically, the spaces are lined by single layers of elongated flat cells of the endothelial type. Immediately beneath this is a thin layer of dense connective tissue with varying amounts of adult type fat cells. Moderate perivascular infiltration by lymphocytes is present. The fluid content presents a milky appearance and the specific gravity is 1.020. Wet smears reveal numerous small, and a few large, refractile globules. The latter are stained bright red and the former pink with Scharlach R. A stained smear reveals rare lymphocytes.

PATHOLOGIC DIAGNOSIS: Cystic lymphangioma with chylous content.

CLASSIFICATION

The classification of cysts found within the mesentery, the omentum, and the group situated retroperitoneally without reference to either mesentery or omentum, have long been considered separately and a different classification for each group has developed. Since the mesenteric and omental varieties are in reality also retroperitoneal because they are situated in anterior extensions or inclusions of this space, Lahey[5] grouped them all together and proposed a new classification. This classification has been criticized by some writers, but it appears to be the most adequate and satisfactory yet proposed. Cysts which are part of adult retroperitoneal organs are not included in this general group. Lahey's classification follows:

RETROPERITONEAL CYSTS

A. Classification according to location:
   1. Omental
   2. Mesenteric
   3. True retroperitoneal (not omental or mesenteric)

B. Classification according to type:
   1. Wolffian cysts, arising from persistent remnants of any part of the early urogenital system.
   2. Lymphatic or chylous cysts, arising from developmental or obstructive phenomena of the retroperitoneal lymphatic system.
   3. Dermoid cysts, resulting from imperfect closure of the abdominal plates, strayed genital cells or supernumerary ovaries. This group includes entercystomas.
   4. Mesocolic cysts, formed from pockets of peritoneum left between the opposed seerous surfaces of the mesentery

and perietal peritoneum in the early rotation of the colon (described by Handfield-Jones).

5. Parasitic and inflammatory cysts.
6. Traumatic blood cysts.

The cyst reported belongs to the second group, the lymphatic or chylous cysts, and was situated retroperitoneally without reference to the mesentery or omentum. These are the most infrequent type of retroperitoneal chylous cysts, the majority of which are situated within the folds of the mesentery anywhere throughout its length.

Pathogenisis

Retroperitoneal chylous cysts are benign. They are usually multilocular and vary in size, often attaining large dimensions. The cavities are thin-walled and smooth and do not as a rule communicate with each other. The walls consist microscopically of connective tissue, with or without an epithelial lining, which, when present, is a single layer of low cuboidal cells. Perivascular lymphocytic infiltration is frequent. The fluid content is characteristically milky in appearance, alkaline in reaction, has a specific gravity of 1.015 or 1.016, is high in albumin content, with blood cell debris and fat or cholestrin crystals.[6]

The origin of chylous cysts is in dispute. Some believe they result from acquired dilatations of varicosities of adult lymphatics, others that they are congenital or that they arise from infiltration of chyle into an adjacent preformed serous cyst. Rokitansky and Virchow believed them to be due to degeneration of lymph nodes. Parsons suggests that their formation may depend on the as yet unexplained phenomena involving the formation of lymph and upon whether the endothelial cells actively secrete or merely conduct lymph.

Symptoms and Diagnosis

Retroperitoneal chylous cysts are almost never diagnosed clinically, but are found at operation for an indefinite or mistaken diagnosis, or are discovered at necropsy. Many of them produce no symptoms and are often discovered during a routine examination. Occasionally the patient may become aware of the presence of an indefinite mass or of fullness, especially on change of position. Mild discomfort or tenderness, or digestive disturbances, are occasionally present. The mesenteric cysts are more apt to produce digestive symptoms and not infrequently become apparent clinically when adjacent bowel becomes obstructed. Cachexia has rarely been observed.

On examination the cyst is often vaguely or inconstantly palpable, it may present in any portion of the abdomen and is movable, cystic and slightly tender. Laboratory data are useful largely in the elimination of other conditions and are rarely of positive value. Roentgenograms have disclosed calcification of the walls when that rare complication occurs and enabled Carter to make a diagnosis clinically.

Complications

Complications are rarely encountered in the non-mesenteric cysts. Partial calcification within the walls has been observed in three instances.[4] Rupture, volvulus, inflammatory changes, pressure obstructions of adjacent hollow viscera such as bowel or ureter, and hemorrhage are exceedingly rare, although these complications may occur, especially in the mesenteric type. Malignant changes are exceedingly infrequent.

These complications may result in acute abdominal pain, nausea, vomiting, elevation in temperature, pulse and blood counts, with abdominal tenderness and rigidity, and lead to the diagnosis of an acute surgical abdomen. Where the mesenteric cysts produce pressure on adjacent bowel, they present data typical of intestinal obstruction.

Treatment

The treatment of all types of retroperitoneal cysts is surgical unless there are absolute contraindications. The true retroperitoneal type is usually situated in loose areolar tissue and is without pedicle or other attachments, hence can be enucleated easily and the peritoneal defect closed without drainage. The mesenteric cysts, in contrast, are sometimes difficult to enucleate on account of their close association with the mesenteric vessels and intestine and resection of the involved segment of bowel may become necessary. Marsupialization of the cyst or simple aspiration and drainage have been done, but these would seem to be unsatisfactory procedures and rarely necessary. Recovery without complications or recurrence has been the rule.

Summary

Retroperitoneal cysts are distinguished from cysts attached to or part of an adult retroperitoneal structure: they may be situated in the omentum, within the folds of the mesentery, or remain in the retroperitoneal spaces without reference to these structures. Chylous cysts in the last group are exceedingly rare: 19 cases were en-

countered in the literature and an additional case is herewith reported.

These cysts are rarely diagnosed clinically, and are usually found at operation for surgical emergencies of mistaken or undetermined origin, or diagnosed as abdominal tumors of undetermined type and localization. Simple enucleation is usually not difficult and the prognosis is excellent.

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MODERN MANAGEMENT OF PERIPHERAL VASCULAR DISEASES

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ARTERIOSCLEROSIS OBLITERANS

THIS is the commonly seen vascular disease of the aged and the diabetic. Generalized arteriosclerosis exists to a greater or lesser extent. Fibrosis, necrosis, calcification of the media, atheroma of the intima and thrombosis characterize the lesion. The process is presumed to result from degeneration of the major arteries with age, wear and tear. Disturbances of the lipoid metabolism are thought to accelerate the development of the atheroma. Arteriosclerosis Obliterans is to be distinguished from Thromboangiitis Obliterans, an inflammatory vascular disease of relatively young males.

Diagnosis: Intermittent claudication or "leg angina" is usually the first symptom of arterial vascular deficiency. Claudication at the arch of the foot suggests arterial deficiency at or above the ankle, claudication at the calf suggests occlusion at or above this level. Claudication, developing suddenly after an attack of severe pain in the leg, suggests that sudden arterial occlusion has occurred as the result of embolism or thrombosis. We record the number of blocks the patient can walk or the time he can walk at 120 steps a minute before claudication develops. Confronted with a patient 50 years of age or over, who complains of intermittent claudication, we suspect arteriosclerosis obliterans and perform the following tests: palpation of the major arteries, elevation-dependency, oscillometry. Abnormal pallor on elevation, rubor and delayed filling of the veins on dependency indicate occlusive arterial disease. We have found the return of color and vein filling time the most valuable indicator of improvement under treatment. Ordinarily color returns and the veins fill within 10 seconds when an extremity is placed in a dependent position after elevation. Oscillometry has been used by us in the diagnosis of peripheral vascular diseases for years. By taking oscillometric readings of the lower extremities of all patients over 50 years of age we have often detected early vascular insufficiency before other symptoms and signs have developed.

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Presented at De Paul Hospital as part of the Spring Clinic of the Norfolk County Medical Society.

And treatment is remarkably effective when instituted at this early stage. The oscillometer determines principally the status of the main arterial channels. Further studies include: a glucose-tolerance test, blood cholesterol and cholesterol esters, an electrocardiogram, soft tissue x-ray for evidence of calcification of arteries. Often it is desirable to know the status of the minute arterial pathways and the collateral circulation. We have found the histamine flare test and the fluorescein test of value for this purpose and perform them as follows:

The Histamine Test.

Use ink or a skin pencil to place horizontal marks at the points to be tested, above the ankle, on the mid-lower leg, below the knee and on the lower thigh. By means of a syringe and needle place a drop of histamine acid phosphate (2.75 mg. per c.c.) at each of the levels indicated. Prick the skin about ten times through each drop of histamine just as one would perform a vaccination. Make observations at 2½, 5 and 10 minute periods. The development of a wheal is the main feature of the test; but a flare or zone of erythema develops around the wheal when cutaneous circulation is good. In normal subjects the wheal begins to appear in 2½ minutes, and is well developed in 10 minutes.

The Fluorescein Test.

The patient lies on his back in a warm darkened room. Scratch the leg at various levels with a needle or the back of a scalpel. Inject 3 c.c. of 20 per cent fluorescein intravenously. Expose the extremity to ultra-violet light, using a special Wood's filter. Note the time at which greenish yellow fluorescence occurs in the hitherto dark purple scratch marks. Fluorescence should occur at the foot level in 30 to 40 seconds, but in arteriosclerosis obliterans and thromboangiitis obliterans may be delayed for 1 to 10 minutes or more.

We habitually perform the histamine or fluorescein test before leg amputations to determine the level at which we may expect primary skin healing.

Beverly Smith has recently reported on the use of radioactive phosphorus in the study of peripheral vascular disease. We have had no experience with the method.

Therapy: The mainstays of treatment remain unchanged, low fat, low cholesterol diet, abstinence from tobacco, tissue extract parentally, Buerger's exercises, intermittent pressure and suction, intermittent venous occlusion, sympathectomy. The low fat, low

chloresterol diet is particularly applicable to the obese patient who has an elevated blood cholesterol. Tobacco causes some arteriolar constriction and thus is definitely contraindicated when the arterial supply is already diminished. Tissue extracts (depropanex) do not per se influence the course of the disease, but given in doses of 3 c.c. every day or every other day may double or triple the distance that the patient can walk before intermittent claudication occurs. The cause of the pain of intermittent claudication is presumed to be a substance elaborated by abnormal metabolism in the ischemic muscles. Tissue extract is thought to act by improving muscle metabolism. The results of therapy in arteriosclerosis obliterans will depend largely upon the earliness with which the diagnosis is made and treatment begun. Notwithstanding some of the articles which have appeared in the literature I can say unequivocally that the leg pump, or intermittent pressure and suction apparatus, used before advanced pre-gangrenous changes have occurred, and continued long enough (3 to 6 months) offers an excellent prognosis. We now have in our files many cases in which collateral circulation was established by this method, and in which peripheral vascular insufficiency never again occurred. Our results with intermittent venous compression have been unpredictable and generally poor. However, an occasional patient seems to respond well, reporting lessened pain and increase in claudication time. The apparatus has the advantage of permitting home treatment in sessions of several hours two or three times daily.

Lumbar sympathectomy for arteriosclerosis obliterans has received a new impetus since it has been recognized that the capacity for arteriolar dilatation may be considerable, the small artery not being generally affected by the arteriosclerotic process. After sympathectomy one may expect a warm dry extremity, with skin better able to withstand minor trauma. Mild rest pain may be relieved and the claudication time may be increased. Sympathectomy should preferably be performed only on patients who are less than 50 years of age and who have a good vasomotor response.

Walking: Exercise usually causes dilatation of arteries, although arteriospasm may occur on exercise in a few cases of organic arterial disease. We regularly encourage our patients with arteriosclerosis obliterans, who do not have coronary insufficiency, to walk several times daily, trying slowly to increase the distance, always stopping short of the occurrence of intermittent claudication. We try to teach a "shuffle gait," flat footed and straight legged. Using this gait avoids repeated dorsiflexion of the foot with resultant compression of the deep calf vessels. A fair percentage of patients

has observed a definite improvement in claudication time by using this manner of walking.

We have discussed methods of treatment of arteriosclerosis obliterans with which we have had experience. New treatments are constantly being tried. Alcohol injection of the lumbar sympathetics has been reported by Lilly. The procedure relieved his patients of pain and gained time for the development of collateral circulation. Katz is using ether intravenously at the Touro Infirmary and Marine Hospital in New Orleans. Five to 10 c.c. of pure chemical ether in 200 c.c. of sterile distilled water are given intravenously daily for several weeks. The method is still in the experimental stage. Intravenous sulphur compounds (Tetrathione) have been advocated in the theoretical hopes that they improve the oxygen-carrying power and reduce the viscosity of the blood. We have been unable to evaluate this form of therapy.

Thromboangiitis Obliterans, or Buerger's Disease

This is a disease of young men, characterized by inflammation of the medium-sized and smaller arteries and veins of the extremities, accompanied by marked vascular spasm and episodes of thrombosis. The disease usually begins before the age of 40 and a diagnosis of thromboangiitis obliterans, when symptoms are initiated at a later age, is open to question. The vessels most commonly affected are the anterior and posterior tibial, radial and ulnar arteries. Large arteries, such as the femoral and brachial, are affected only late in the disease. The inflammation of the vessels is patchy and segmented, leaving areas of normal vessels between the affected portions. A superficial thrombophlebitis occurs at some stage of the disease in about 40 per cent of cases. The onset of the disease is usually insidious, for neither the arteritis, the associated phlebitis, or the superficial thrombophlebitis, produces significant symptoms until episodes of thrombosis have diminished the blood supply to the extremity. Then the cardinal symptom, intermittent claudication, develops. The pain of intermittent claudication and the more severe pain of ischemic neuritis dominate the symptomatology of thromboangiitis obliterans.

The cause of thromboangiitis obliterans is still unknown. No specific treatment has been discovered. But the pathologic picture is better understood, and we have learned to use the treatments at our disposal more rationally. The disease produces organic, usually complete and permanent, occlusion of the segment of vessel affected. This occlusion, particularly of the arteries, leads to the shunting of the arterial flow through uninvolved anastomatic ves-

sels and, more significantly, is followed by extensive development and enlargement of collateral circulation. Therapeutically we endeavor to encourage the development of this collateral circulation, relieve the coexisting vascular spasm and reduce the thrombotic tendency. Smoking produces vasoconstriction in the majority of persons, whether or not they have vascular disease, and smoking has a particularly pernicious effect in Buerger's disease. Smoking is therefore to be completely and permanently given up. The possibility of disaster resulting from even an occasional smoke must be emphasized. Next in importance is sympathectomy, lumbar sympathetic ganglionectomy for the lower extremities and cervicothoracic sympathetic ganglionectomy for the upper extremities. This provides maximum vasodilatation at any stage of the disease, increases the effectiveness of the developing collateral circulation, produces a warm dry extremity, makes possible the effective use of mechanical means of stimulating the development of collaterals. Neither the paevex apparatus or the rhythmic constrictor can be effective in the presence of vasoconstriction, and vascular spasm may actually be aggravated by the paevex machine, especially in acute phases of the disease. Our routine, therefore, is first to perform sympathectomy, then prescribe mechanical means of increasing circulation, paevex therapy or the rhythmic constrictor, and Buerger's exercises.

While this course of mechanical therapy is in progress we usually carry out a parallel course of intravenous infusions, 300 c.c. of 5 per cent sodium chloride, in the hope of reducing the thrombotic tendency until ultimate cessation of development of new occlusive arterial lesions occurs. When the diagnosis of thromboangiitis obliterans is made early and the above plan is followed, the prognosis as to loss of limb is often very good. Some claudication may persist for life but not even a digit need be lost. We continue to use tissue extract (depropanex) for the claudication of these chronic cases and some report improvement. When severe arterial insufficiency, as the result of the extensive organic occlusion of arteries, exists treatment has little to offer. Again, as in many diseases, the diagnosis must be made early in Buerger's disease if treatment is to be effective.

During exacerbation of the disease and when rest pain, pain of ulceration and gangrene are present, the patient is hospitalized and treated with typhoid injections after the method of Mayo Clinic. The usual course is an injection every other day until 8 to 12 injections are given. Relief of pain and healing of ulceration usually occur. Sympathectomy and the program previously described may then be carried out.

One would assume that, in a disease characterized by episodes of thrombosis, the new anticoagulants, heparin and dicoumarin would prove especially valuable. Actually it would be necessary to continue anticoagulant therapy for months or years, as the disease is usually chronic and the episodes of thrombosis occur at widely separated intervals of time. Heparin and dicoumarin may be of value over short periods of time during acute phases of the disease and are definitely indicated should sudden extensive arterial occlusion occur, in order to prevent clotting of blood distal to the occluded segment.

Arterial Embolism

This is not an uncommon occurrence and I now have been called to see a number of such cases. With the exception of clots detached from atheromatous plaques in the aorta, all arterial emboli originate from thrombi within the heart. The most outstanding element of the previous history is cardiac disease attended by fibrillation. The cardinal symptoms are sudden pain in a lower extremity, followed by pallor and coldness of the part, collapse of the cutaneous veins, subsequent cyanotic discoloration of the part, with absence of peripheral pulsation and loss of sensation. Abrupt pain may be entirely absent, numbness, coldness and tingling being the only initial symptoms. The most frequent site of localization is the lower femoral artery. The next common site is at the branching of the common femoral and profunda femoris. The point of lodgment is usually where the major vessel branches. Extreme tenderness over the region of occlusion is common. The exact point of lodgment is determined by noting the level at which arterial pulsation is still present and the level at which arterial pulsation is no longer palpable. Oscillometric readings help and changes in skin temperature help. The line at which the temperature of the skin changes from low to normal is located just above the ankle in occlusion of the popliteal artery and at the junction of lower and middle thirds of the thigh in occlusion at the bifurcation of the femoral artery. When the common iliac artery is occluded the point of skin temperature change is located at about the junction of middle and upper thirds of the thigh.

Patients who are digitalized after coronary disease are especially apt to throw off peripheral emboli.

Treatment of arterial embolism: Treatment may be medical or surgical. The medical treatment consists of opiates for pain, of papaverine hydrochloride 1/6 to 1/2 grain intravenously, repeated every 8 hours, repeated sympathetic novocaine blocks, anticoagu-

lant therapy with heparin and dicoumarin, continuous use of rhythmic constricter. The extremity should be level with the bed or slightly lowered. It should be wrapped loosely in cotton in order to preserve the natural warmth of the extremity. Cooling reduces the metabolism of the leg and thus lessens its need for blood, but it also increases the vascular spasm present. Therefore, unless it is known that irreversible changes have occurred and that amputation is inevitable, cooling should be withheld and, when applied, should be slight only—effected by a few ice bags covered with cloth, and the limb protected further with sheet wadding. Moreover, lumbar sympathetic novocaine injections should be done repeatedly during the cooling phase to offset the spastic effect. The anticoagulant and antispasmodic therapy is extremely important as it is highly desirable to prevent thrombosis of the artery distal to the site of the embolus.

Medical management is indicated when the embolism is low, such as in the popliteal artery, when the case is seen later than 10 hours after the embolic obstruction, and when it is impossible to decide whether one is dealing with embolism or thrombosis.

The surgical treatment consists in embolectomy. About one half of the patients having embolectomy within the first 10 hours leave the hospital with restored circulation. Embolectomy is not a formidable procedure. It usually consists of exposing the lower or upper femoral artery, performing arteriotomy, extracting the embolic mass and the more peripheral thrombus and approximating the arterial incision edge to edge with the intima everted by fine arterial silk sutures. Anticoagulation therapy with heparin and dicoumarin and repeated novocaine blocks should precede and follow arteriotomy. The heparin therapy should be started as soon as the diagnosis of arterial embolism has been made. This is done to prevent a thrombus from occurring within the arterial system distal to the embolus until the removal of the embolus can be carried out. After the embolus and peripheral thrombus have been removed heparin may be injected intraarterially through the arteriotomy opening.

Arterial Thrombosis

Arterial occlusion may occur as the result of local thrombosis rather than of embolism. Generalized infection and bacterial toxins may set up an inflammation of the artery which leads to thrombosis. Trauma may cause acute arterial thrombosis. In 1940 we described two such cases, one developing during the use of an air hammer and the other from the use of a leg operated tractor. Arterio-

sclerosis may result in thrombosis on the roughened intima or on atheromatous plaques. Treatment of arterial thrombosis is identical with the medical treatment of arterial embolism.

Differential Diagnosis Between Arterial Obstruction Due to Embolism and Due to Thrombosis

The cause of sudden arterial occlusion may not always be easy to determine. When heart disease is present, and particularly if it is associated with fibrillation or if a coronary infarction is known to have occurred, the sudden occlusion may usually be attributed to embolism. This is especially so if there is no evidence of long-standing peripheral arterial disease. When sudden arterial obstruction occurs in the presence of evidence of thromboangiitis obliterans and arteriosclerotic vascular deficiency, the arterial occlusion can safely be attributed to thrombosis occurring as a part of the above diseases. Insidious or slowly developing arterial occlusion is practically always due to local thrombosis rather than to embolism.

The Injured or Severed Artery

When the main artery of an extremity has been severed it may be possible to repair the damage rather than to ligate the injured artery. The advent of anti-coagulation therapy has increased the percentage of successful arterial sutures. A non-suture method of arterial anastomosis has been described by Blakemore using vitallium tubes. If a defect is present a segment of vein is used to bridge the area, for instance, a segment of saphenous vein. The ends of the vein are turned back as cuffs over the vitallium bands. The reports from the theatres of operation in World War II have not indicated that this procedure is more successful than other methods of arterial suture.

An equally successful method seems to be the use of the soluble rod as described by Captain Sidney Smith. Dextrose is heated until slightly caramelized and then poured into a sterile rubber tube, the inside diameter of which approximates the diameter of the artery to be repaired. The filled tube is then dropped into ether for a few minutes. The rubber softens and swells, permitting the rod to be slipped out of the rubber mold with ease. The rod is then coated with paraffin. This soluble rod is then inserted one half of its length into one end of the artery and the other half into the other end and arterial suture is performed. A warm saline solution is then applied over the site of the anastomosis to melt the protective paraffin coating. The rod goes into the solution and the pulsating blood is noted in a minute or so.

Varicose Veins

High saphenous ligation remains the best method of therapy for varicose veins. However, it is not a cure-all and recurrences occur no matter how thorough the operative procedure is. This is to be

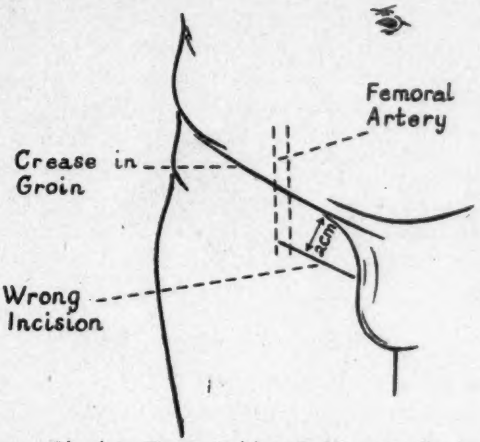

Fig. 1. ***Saphenous Ligation, Wrong Incision***—Incision, usually described 2 cm. below crease in groin. Wrong. Necessitates dissection high up under upper flap and invariably leads to the missing of some branches.

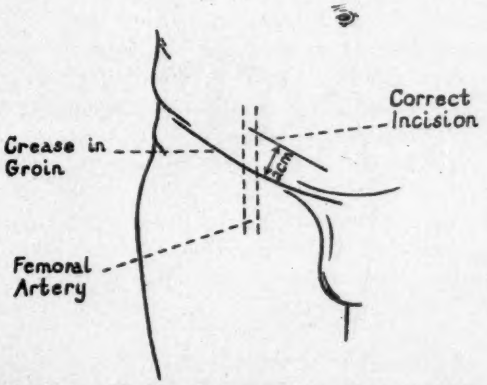

Fig. 2. ***High Saphenous Ligation, Correct Incision***—Correct incision 1 to several cm. above crease in groin, depending on weight of patient.

presumed when one realizes that communicating veins which were not incompetent at the time of the initial surgery may at subsequent date become incompetent, and that tiny collateral channels which were invisible can dilate to form real communications at a later date. Experience has taught us several pertinent points.

1. We are ligating higher and higher. Our incision is never below the crease in the groin, and in an obese patient the incision is 1 to 3 cm. above the crease in the groin. Thus our incision is on the abdomen rather than on the leg. This brings us down on the

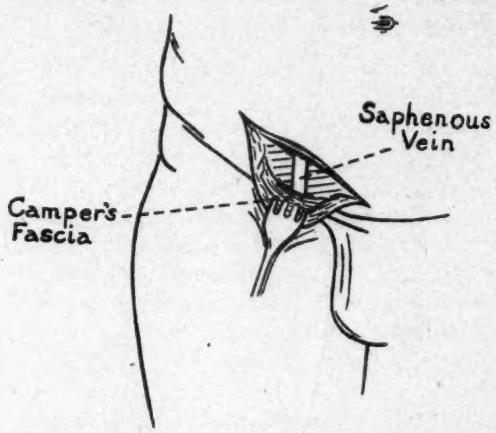

Fig. 3. *High Saphenous Ligation*—Lower flap retracted. Dissection superficial to Camper's fascia to below crease in groin. Camper's fascia incised, and saphenous, located in lowermost portion of wound, and divided.

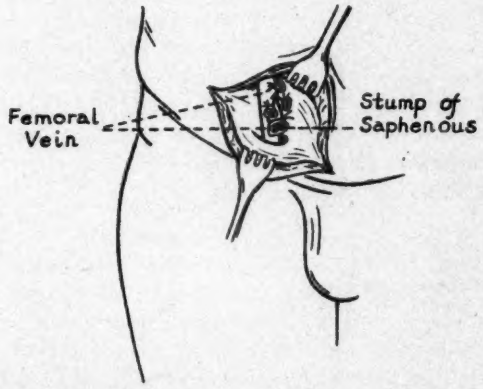

Fig. 4. *High Saphenous Ligation*—Note that the incision described enables careful dissection of all branches under good exposure. The femorosaphenous junction is in the center of the wound.

saphenous at the femorosaphenous junction and enables us to visualize well all the branches, particularly the epigastric branch which often is not found when the incision is lower.

2. We carefully dissect out the femorosaphenous junction so as

to locate and divide any unusual branches. We have come to realize that it is easy to ligate and divide the saphenous vein, but hard to cure varicose veins.

3. We do not inject at the time of our saphenous ligation. This is omitted because occasionally the amount of solution injected will produce a thrombophlebitis of greater severity than necessary, causing the patient to go to bed and causing undue edema. Our injections are given at the office when the stitches are removed and at other visits. We still use sodium-morrhuate, or if allergy to this develops, we use soricin or etalate or sylnasol.

4. We insist that our patient get up and walk every half hour after the operation is performed on the operative day and remain at his usual occupation during the subsequent days. Figures 1 to 4 indicate our technic for high saphenous ligation. The incision is above the crease in the groin.

Varicose Veins and Pregnancy

We prefer not to perform high ligation of the saphenous during pregnancy. The marked downward venous pressure due to the pelvic congestion and pressure on the iliac vessels by the gravid uterus tend to offset the value of the operation. Should an ilio-femoral thrombophlebitis occur in the postpartum period most of the venous return has been interfered with. The venous congestion, many of the dilated venules and even some of the varicosities will disappear after the delivery. Where abnormally large varicosities are present and the patient is seen in very early pregnancy we have in exceptional instances performed high saphenous ligations with no untoward results. However, recently we had a fatal embolism occur in a woman who had a sterilization procedure on the fifth postpartum day and a saphenous ligation on the twelfth postpartum day. There was no way of telling whether the embolism resulted from the parturition, the laparotomy or the saphenous ligation. This is the only fatal embolism we have had in several thousand vein ligations.

The Ambulatory Suture Ligature Method of Treating Varicose Veins

The ambulatory suture ligature method of treating varicose veins is particularly suitable to the treatment of varicosities during pregnancy. The weight of the downward course of blood is interrupted. The varicosities are taken care of effectively while the contributing cause of the downward venous pressure, that is the pregnancy, is in existence. And, during pregnancy, thrombosis occurs

regularly distal to the suture ligature, or is readily effected by injections.

This subcutaneous method of placing the ligature, applicable to ambulatory patients, was first described by Goldstone in the *British Medical Journal*. The technic is simple—a curved needle passes through the skin under the vein and out through the skin of the opposite side. A straight needle is substituted for the curved one and re-enters the skin to pass over the vein and emerge at the original point of entry of the curved needle. The two ends of the catgut are then tied together forming a true ligature around the vein. The knot is cut close and drops subcutaneously out of sight. The point selected for the ligature is the uppermost large dilatation of the saphenous. The method is a simple office procedure.

The literature is full of modifications of the high saphenous ligation procedure for varicose veins. Thus Hodge, Grimson and Schiebel have recently advocated high saphenous ligation plus the old-fashioned vein-stripping and evulsion procedure.

Migratory Phlebitis

Very little progress has been made in our knowledge of migratory phlebitis. We recently obtained an excellent result using dicoumarin. A heparin tolerance test should be done in all such cases. The people are probably hyporeactors. Typhoid vaccine intravenously has helped some cases. Migratory phlebitis in the male may be an early manifestation of Buerger's disease.

Femoral Thrombophlebitis and Phlebothrombosis

This subject is discussed by me in the *American Journal of Surgery* for December, 1945. Our ideas on this subject have changed very little since the writing of that paper, although we are using dicoumarin more extensively. Our present principles are as follows:

1. Acute obstructive iliofemoral thrombophlebitis or milk leg without pulmonary embolism. Therapy—repeated novocaine blocks of the lumbar sympathetics and dicoumarin by mouth.

2. Acute iliofemoral thrombophlebitis with pulmonary embolism. Therapy—bilateral femoral exploration, embolectomy, and ligation. We usually ligate and divide the femoral above the profunda on the affected side, ligate the superficial femoral (below profunda) on the opposite side.

3. Persistent iliofemoral thrombophlebitis with pulmonary embolism. Therapy—iliac vein ligation.

4. Thrombophlebitis of deep calf veins in absence of embolism. Therapy—dicoumarin by mouth.

5. Obvious ascending phlebothrombosis. Therapy—prophylactic superficial femoral vein ligation.

6. Phlebothrombosis in the aged or the poor risk, even in the absence of pulmonary embolism. Therapy—prophylactic superficial femoral vein ligation.

We do not believe that one is ever justified in watchful waiting and no therapy in thrombophlebitis. We have saved many lives by active intervention i.e., femoral vein embolectomy when embolism threatened or existed.

Vena Caval Ligations

As already indicated, femoral vein ligations have become rather commonplace. Recently vena caval ligations have been advocated for selected instances of advanced iliofemoral thrombophlebitis. Actually vena caval ligations may be less radical than femoral ligations in the presence of bilateral extensive iliofemoral thrombophlebitis and one is more assuredly above the thrombophlebitic process, and there is even less interference with the circulation. At a recent meeting in New Orleans I heard a paper reporting 8 successful vena caval ligations for pelvic thrombophlebitis. Bilateral lumbar sympathectomy was performed simultaneously to prevent lymphedema with good results. Cava and iliac ligations are technically difficult and dangerous and should not be attempted by the inexperienced.

Concomitant Thrombophlebitis in Varicosed Saphenous and Deep Vein Phlebothrombosis

One occasionally sees a patient who has developed spontaneous thrombophlebitis in his varicosed saphenous and who also presents signs of deep vein phlebothrombosis—a positive Homan's sign, calf tenderness. It has been our practice to hospitalize such patients, using anticoagulant therapy to quiet down both processes. When there is reason to believe that the deep vein involvement has healed in situ, we perform the usual high saphenous ligation. Instead of allowing the patient to go home the day of the operation as us customary, we keep him in the hospital an additional four or five days during which time we continue anticoagulant therapy. To date we have not had embolism with the above program nor have we subsequently had to tie the femoral. By avoiding tying the femoral the patient has no subsequent leg edema.

Chronic Thrombophlebitic Edema and Induration

In mild cases of edema the lymph circulation may be restored to competency by fluid and salt restriction, diuretics and elastic support. Often novocaine injections of the lumbar sympathetics will help even the chronic case. Lumbar sympathectomy offers the patient his best means of permanent relief of such edema. Lumbar sympathectomy is also of value in relieving the edema due to mild recurrent episodes of superficial thrombophlebitis in varicose veins. If the soft parts are contracted, indurated and fixed, very little benefit can be expected from sympathectomy. Removal of the scarred fibrotic subcutaneous tissue and fascia is the procedure of choice. The above remarks are not applicable to lymphedema praecox, lipedema and other types of edema in which the vasoconstrictor factor does not exist. Sympathectomy is of no benefit in these cases.

Treatment of the sequelae of deep vein thrombosis has taken a peculiar turn in the hands of Drs. Buxton and Coller of Ann Arbor, Michigan. These authors report a group of cases of recurrent leg ulcers due to deep vein thrombosis treated by femoral and vena caval ligations. They report a high incidence of healing of the static ulcers. Femoral and vena caval ligations for the relief of the sequelae of deep vein thrombosis must certainly be said to still be in the experimental stage.

Sympathetic Nerve Block

Novocaine block of the sympathetics is now a well-established procedure. The technic is readily acquired and should be learned by every practitioner. The best results are obtained when the procedure is performed as soon as the diagnosis is made, of acute arterial thrombosis, of acute arterial embolism, or of acute iliofemoral thrombophlebitis. Novocaine block of the sympathetics is also valuable preceding and following vascular surgery and in chronic lymphedema and in causalgia.

Lumbar Sympathectomy

Lumbar sympathectomy, or surgical removal of the 2nd, 3rd, and 4th lumbar sympathectics has also emerged from the realm of speculation and has become a valuable method of treating vascular disease. Lumbar sympathectomy is indicated when considerable vasospasm exists in arteriosclerotic vascular deficiency, in Buerger's disease and in Raynaud's disease, and as an adjuvant to the vascular surgery of the lower extremities. Lumbar sympathectomy helps in the management of wounds of the extremities involving the blood vessels and in the therapy of lymphedema which has resulted from

deep vein thrombosis or recurring episodes of thrombophlebitis in varicose veins.

We use a retroperitoneal muscle splitting approach similar to that used for high ureteral calculi. The procedure is not difficult. Convalescence is rapid, the patient leaving the hospital on the fifth to seventh day.

Anti-Coagulant Therapy

It is apparent from the numerous times that references have been made to heparin and dicoumarin, that we feel that anti-coagulant therapy has an important role in the management of vascular disease. Mention has been made of the value of anti-coagulant therapy in arterial embolism, arterial thrombosis and venous thrombosis. Heparin will raise the clotting time within an hour or two. Dicoumarin orally has a latent period of 48 hours. It is, therefore, practical to combine the two anti-coagulant agents at the onset of therapy. Heparin is given intravenously immediately on making the diagnosis of acute arterial obstruction, acute phlebothrombosis, acute thrombophlebitis, or pulmonary embolism. The best method of administering heparin is to add the heparin to the intravenous saline drip, in the proportion of 20 units of heparin to 1 c.c. of saline. This usually means adding two 10 c.c. vials, 20,000 units, or 200 mg., to 1,000 c.c. of saline. The saline drip is set to run at 25 to 30 drops per minute. An alternate method is to give repeated intravenous injections every 3 hours of 50 mg. (5000 units) of heparin. Should the undiluted heparin cause untoward reaction, each 50 mg. may be given in 250 c.c. of saline. The heparin is discontinued 24 to 36 hours after the initial dose of dicoumarin.

The dicoumarin therapy is initiated with 300 mg. of dicoumarin on the first day. Fifty mg. to 100 mg. of dicoumarin are then given orally daily to maintain the prothrombin time at about 20 to 30 per cent of normal for 2 to 3 weeks. Hykinon 60 mg. and whole blood are given if the prothrombin time drops below 20 per cent or if hemorrhage occurs. We have used anti-coagulants, especially dicoumarin, more or less routinely for over 2 years with no untoward result. However, all of our cases are hospitalized and the therapy carefully controlled. A daily prothrombin time is taken and the dose of dicoumarin is predicated on the result of this daily test. Even if one doubts the efficacy of anti-coagulant therapy in preventing embolism, it should be part of the therapy of all peripheral vascular thrombotic problems—for no other reason than to prevent extension of thrombosis into more of the vascular tree.

Amputation

I should like to touch briefly on the subject of leg amputation. We prefer the Pearl modification of the Callender procedure for all lower thigh amputations. In this procedure no tourniquet is used, the vascular supply is controlled at the outset, the amputation is through tendons, not muscles. The skin invariably heals by primary intention and abundant soft tissue is present over the stump. The patient may usually leave the hospital 10 days after the amputation without an open wound. This is in marked contrast to the guillotine procedure or to the flap procedure with drainage.

Another pet hobby of ours is prophylactic femoral vein ligation in all leg amputations in the aged. We simply expose the femoral vein in Scarpa's triangle, ligate and divide it distal to the profunda before beginning the amputation. Absorption of septic products is thus prevented, and what is more important, all danger of postoperative embolism from the amputation stump is obviated. Death immediately following amputation in these patients is almost always due to detachment of an ascending thrombus initiated in the popliteal vein at the site of amputation. One has only to perform a few femoral ligations prior to leg amputation to realize how unusually smooth the convalescence is in such instances.

We have previously referred to the value of the histamine test before leg amputation. It will tell you at what level primary skin union may be expected.

Refrigeration Anesthesia

Refrigeration anesthesia is a valuable method for the debilitated hopeless risk, who could stand none of the orthodox methods of anesthesia. Refrigeration anesthesia is adequate for amputation only when the leg is elevated and a tourniquet applied and then the leg lowered and refrigeration applied to well above the level of the tourniquet. This invariably slows the healing of the stump. I feel that this method should be reserved for cases it was intended for, those who could not stand any other type of anesthesia.

Refrigeration is of great value in arterial occlusion with obvious irreversible changes and impending gangrene. Refrigeration then removes the cause for haste and permits deferring the amputation until the patient's condition has improved. The same usefulness of refrigeration anesthesia occurs in the severely infected extremity with inevitable amputation. As the limb becomes refrigerated sepsis and absorption cease and the general condition of the patient improves, permitting amputation at optional time.

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VOLVULUS NEONATORUM

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THE occurrence of volvulus neonatorum and congenital duodenal obstruction has been noted sporadically in the literature for several years. However, it still remains a rarity. In a review of the literature in 1914, Weible reported 67 cases.[1] In another review in 1922, Davis and Poynter[2] presented 392 cases of congenital intestinal obstruction of all parts of the intestinal tract above the anus, proved either by autopsy or operation. Of these, only two patients recovered, a mortality of 99.5 per cent. Recently, Swenson and Ladd[3] reported 21 cases of intrinsic duodenal obstruction with 13 recoveries after operation, and 30 cases of extrinsic obstruction with 26 recoveries. According to Mellins and Milman[4] "this striking decrease in mortality can be, to a considerable extent, attributed to the early roentgenographic diagnosis of these developmental abnormalities." Swenson and Ladd[3] emphasized that "not only is the mortality in a given case directly related to the promptness of diagnosis and treatment, but the period of recovery and convalescence in the hospital is decreased." Infants are a better surgical risk during the first two days of life than a week later, because of better electrolytic balance. However, with proper care in maintaining this balance and preventing dehydration and hypoproteinemia, the operation may be delayed, as shown by the case which we will present. Ladd[3] emphasized that perforations may occur during the first two days and, therefore, surgical intervention should take place as soon as possible.

The following case of torsion of the whole mesentery of the small intestine is presented owing to its rarity and other special features of interest. We wish to acknowledge our indebtedness to Dr. Maurice J. Small who referred the case.

Baby J. L., a white male, was born on Aug. 26, 1946, the second sibling of parents aged 27 and 25 years. Labor was uncomplicated, with spontaneous delivery in twenty minutes after arrival of the physician. The child breathed and cried promptly and no resuscitation was required. Six days after birth, he was admitted to the pediatrics service of this hospital with a history of vomiting a greenish material. The emesis occurred 15 minutes to 1½ hours

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after every feeding. No stools were passed until the fifth day, when an enema produced a small "mustard-colored" meconium stool. For 3 days prior to admission, the child had received atropine gr. 1/1000 and elixir phenobarbital, 6 drops every 4 hours, 30 minutes before meals, without apparent benefit. Various formulas had been tried but to no avail.

Physical Examination

On physical examination, the child was found to be moderately dehydrated with a dry, loose, slightly icteric skin. The weight was 5 lbs. 2 ozs. (2424.16 Gm.). The eyes, ears, nose, and throat revealed no abnormality. The heart and lungs were normal. The abdomen was flat, but frequent visible peristaltic waves were plainly seen across the epigastrium. These appeared to move to and fro. No masses or organs were felt. The umbilical cord was still attached, but somewhat desiccated. The genitalia revealed a granulating circumcision wound, but no other abnormality.

Laboratory Examinations

The red blood count was 3,800,000; white blood count, 14,000. The differential count revealed 55 per cent neutrophiles (26 filamented and 29 nonfilamented); 39 per cent lymphocytes; 4 per cent monocytes; and 2 per cent eosinophiles. The hemoglobin was 9.5 Gm. (57 per cent). The urinalysis showed a 2+ albumin; 4 to 8 white blood cells per high power field; the urine was dark yellow in color. The serology was negative by Kahn and Kolmer Wassermann reactions. The bleeding time was 1 minute and coagulation time was 3 minutes.

X-Ray Examinations

The patient was observed fluoroscopically and the barium meal seen to enter the stomach normally. A part of the barium passed through the duodenum but met with some apparent obstruction of an undetermined nature and was regurgitated into the stomach. This happened repeatedly with the barium being seen to enter the duodenum and almost immediately to be regurgitated into the stomach.

Conclusion: Undetermined obstruction of duodenum.

Hospital Course

On admission, the baby was placed on an evaporated milk formula with feedings every 4 hours. The emesis of a greenish vomitus equal to the quantities ingested continued. Occasionally, a feeding would be retained until after the second feeding at which time the stomach would empty. It was thought that all food was being vomited; therefore, feedings by mouth were stopped and sterile water used to keep the mouth moistened. Fluid and electrolyte balance was attempted by the use of glucose and saline interstitially.

On the third day after admission, a nasal tube was passed and barium was placed in the stomach and the patient sent to the x-ray department for fluoroscopic examination and roentgenograms. About 100 c.c. of amino acid solution (amigen, lately called proteolysate) was given intravenously after venesection and placing a cannula. This was followed by 500 c.c. of glucose and saline interstitially; 75 mg. of ascorbic acid and 1 mg. of menadione were given by injection.

Two spontaneous greenish colored stools were produced. Since the stools

had a curd-like appearance, it was felt that there might have been some food passing through the intestine. Atropine gr. 1/1000 and phenobarbital gr. 1/8 were given to see if there were spasm which would be relieved by antispasmodics and sedatives. At the same time, small formula feedings of 5 c.c. every 30 minutes were reinstated.

Two days later, further roentgenograms were made and another fluoroscopic examination was carried out. No barium was seen to go past the second portion of the duodenum and a diagnosis of duodenal obstruction, probably from congenital atresia, was made.

The child was given 30 c.c. of whole blood intramuscularly and more interstitial and rectal fluids, making a total of 1200 c.c. of fluids preoperatively. He was then sent for laporatomy on the tenth day of life.

Observation at Operation and Procedure

The attached umbilical cord was first excised. The abdomen was prepared with tincture of merthiolate, and drapes arranged. A right upper paramedian incision was made; and the rectus abdominis muscle split. On opening the peritoneum, the stomach was found to be dilated; the small intestines were completely deflated, quite flat; and had a bluish cyanosis. The small intestines were delivered through the wound and resembled a mass of "fishworms." Large distended vessels were noted near the ligament of Trietz. At its origin, the mesentery was twisted through one complete circle in a clockwise manner. This torsion was relieved by rotating the intestines in a counterclockwise direction one complete turn. Immediately the intestines filled with gas, and the color soon returned to normal. The intestines were replaced in the abdominal cavity and after a small tear in the mesentery was repaired, the abdominal wall was closed in layers.

Postoperative Course

Except for one episode of gaseous distension on the second postoperative day, the abdomen remained flat after operation. There was a reduction in the amount of vomiting, there being only four such episodes postoperatively. The stools soon became normal in color and number. A transfusion of 50 c.c. of whole blood was given on the second postoperative day. Water and dextrose solution in small amounts, totaling between 200 and 250 c.c. per day were given for three days following operation. Six hours after operation, small feedings of sterile water were started by mouth. These were later changed to dextrose solution 2 per cent, and on the third day the child was placed on a weak formula. Feedings were gradually increased to full formula by the seventh postoperative day. The wound was dressed on the eighth postoperative day and all black silk sutures were removed. A small amount of infection was present around the two sutures at the lower portion of the incision. The patient was discharged on the eleventh postoperative day at which time he weighed 6 lbs. and 6¾ ozs. (3193.6 Gm.).

Discussion

Volvulus neonatorum is caused by certain mechanical factors resulting from abnormalities of embryologic and anatomic development. During embryonic growth the intestines rotate upon the superior mesenteric artery using[5] it as an axis. The primary loop

of primitive bowel which leaves the abdominal cavity at the third week of intrauterine life consists of a descending and an ascending limb. From the former is derived the jejunum and most of the ileum; and from the latter the remainder of the ileum, cecum, appendix, ascending colon, and part of the transverse colon. The primitive bowel stalk contains the vitello-intestinal duct which springs from the free border of the bowel at the apex of the loop. It passes to the yolk vesicle and superior mesenteric artery, traversing the entire length of the dorsal mesentery. The only attachment to the posterior abdominal wall at this stage is a narrow band of mesentery between the two loops at their points of origin called the "duodenocolic isthmus."

With degeneration of the yolk stalk, and vitello-intestinal duct, enlargement of the abdominal cavity, and less traction on the mesentery, the intestine is drawn back into the abdominal cavity. The descending limb is now too large to occupy a position in the midline and is thrown into coils and loops, some of which are transverse and others oblique to the axis of the abdomen. The first coils lie on the right side of the superior mesenteric artery, but are gradually pushed to the left and posterior portion of the abdominal cavity by succeeding coils. The cecum is last to enter and lies near the umbilicus and anterior to the superior mesenteric artery and small intestines.

Rotation now begins and takes place on the axis of the superior mesenteric artery at the duodenocolic isthmus through an arc of 180° in a counterclockwise direction. The artery also undergoes rotation. The mesentery is shortened and more horizontally placed in many of these cases. If complete peritoneal fusion does not occur at this stage, volvulus may result.

As Skinner[6] has so well pointed out, "a volvulus such as this does not connote obstruction of the bowels with paralysis resulting from the primary twist, namely, interference with the circulation in the mesenteric vessels. The symptoms, therefore, are not those of acute obstruction (as from a band kind or internal hernia) nor those of a sudden mesenteric thrombosis, but are of an intermediate kind."

The symptoms of persistent vomiting with bile in the vomitus, absence of stools except for meconium stools the first days, and absence of bile in the stool should lead one to suspect an obstruction below the ampulla of Vater. The physical findings of distention of the upper abdomen and flattening of the lower portion (not present in this case but reported by Neimeier),[1] and visible active peristalsis in the upper abdomen should bring obstruction to one's attention.

It is important to note, as Saunders and Lindner[7] have previously stressed, that children with true hypertrophic pyloric stenosis do not vomit until the seventh to ninth day at the earliest, while patients wih congenital duodenal or other upper bowel obstruction vomit within the first twenty-four hours. Reverse peristalsis in the upper abdomen and rapid formation of ketone bodies is also indicative of high bowel obstruction.

The use of roentgenologic methods of diagnosis, by means of contrast media such as barium sulfate or plain roentgenograms with air as the contrast substance, should be made early. Cohen[8] has stated that the latter will suffice in most cases since the swallowed air will fill the bowel above the site of the obstruction. Several authors have pointed out the dangers of the infant aspirating the barium when vomiting occurs, with a severe pneumonia resulting. However, if a small quantity of barium is used and it is made into a thin mixture which can later be removed by aspirating it through a nasal tube, we believe there is little danger.

When roentgenograms reveal an obstruction, surgery should be performed, with the least possible delay.

Summary

1. A case of volvulus neonatorum is presented. 2. Vomiting is an early and persistent symptom and should arouse suspicion of high intestinal obstruction. 3. Roentgenograms with or without contrast media should be used to verify clinical suspicions. 4. As soon as the diagnosis can be established and fluid balance regulated, these cases should be subjected to surgery.

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MANAGEMENT OF DISEASES OF THE PANCREAS

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THE pancreas has always received far too little recognition and consideration in the field of medicine and surgery.

Physiologists have recognized its important role in the digestive process, but when disease of the pancreas was established, the physician and surgeon have approached the problem with an attitude of defeatism.

Within the past decade in some of our clinics, the pancreas is beginning to receive more attention from the scientific medical investigation and in desperation some of the cases have been referred to the surgeon with an increasing, favorable prognosis. The indifference toward pancreatic disease has been due in the past to (1) lack of a satisfactory medical regime, and (2) lack of proven surgical procedures with any degree of assurance of cure or relief.

The location of the pancreas in a deep retroperitoneal position on a level with the first and second lumbar vertebrae make it less available to the surgeon and, therefore, less of a temptation for unproven surgical procedures. It extends from the duodenum, in whose loop the head of the organ is molded, to the spleen on the opposite side. Its proximity to the vena cava and portal tributaries lying just behind and adjacent to the head, the body crossing the abdominal aorta, the renal vessels, left kidney and suprarenal gland makes the approach difficult and hazardous. The tail is in close relation to the spleen and the splenic artery passes along the upper surface of the pancreas through a well defined groove or even a tunnel. It, too, is a vascular problem of no small proportions.

The blood supply to the pancreas itself comes from the splenic, the superior and inferior pancreaticoduodenal arteries. The lymphatic system is rich and drains mostly into the nodes at the head of the pancreas and duodenum and to the pre-aortic area. The solar plexus and vagus furnish the nerve supply.

Histologically, the pancreas is classified as a compound, racemose gland. It is loose in texture and has no capsule. Each lobule is one of the ultimate ramifications of the main duct which is filled with secreting cells that have two distinct zones. The outer zone is probably responsible for the secretion of the pancreatic digestive juices.

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Toward the center the cells become highly specialized, are not connected with the duct system, and are known as the Islands of Langerhans which are richly vascular and which supply the internal secretion of the pancreas—insulin. The main excretory duct of the pancreas is the Duct of Wirsung which traverses the entire length of the gland and opens into the Ampulla of Vater, most usually in association with the common bile duct. An accessory Duct of Santorini is usually present and communicates between the Duct of Wirsung and the posterior wall of the duodenum about 2 cm. above the Ampulla of Vater. In the surgical consideration of pancreatic diseases with which this paper is concerned, certain anomalies of the pancreas should be mentioned.

The Duct of Santorini often atrophies and serves no function at all; yet, Mayo-Robson and Sir Berkley Moynihan[1] found it to be the primary excretory duct in 10 per cent of a large series of patients observed by them.

Opie, Mann and Giorondo,[2] working independently, found that the common bile duct and Duct of Wirsung entered the duodenum through a common orifice in 65 per cent of 200 autopsies and through separate openings in the remaining 35 per cent.

There is described by Hyrtle a condition known as Pancreas Divisum in which there are two separate functioning glands, another in which the tail is bifid, one known as Annular Pancreas in which the gland is seen as a band or ring enveloping the second portion of the duodenum. It is described as two arms of normal pancreatic tissue, extending anteriorly and posteriorly from the head of the pancreas and embracing partially or completely the duodenum. Forty-nine such cases have been collected and reported by Lehman.[3]

Symptoms arising from such a condition are those of duodenal obstruction which would arise by the contracting, diseased arms of the pancreas and which would also obstruct the opening of the common bile duct resulting in jaundice, nausea, vomiting, pain, etc. There is no clinical characteristic syndrome of annular pancreas; however, radiographic evidence of partial obstruction of the second portion of the duodenum should direct one's attention to such a possibility. There is an aberrant or accessory pancreas described by Krieg[4] who reviewed the literature and reported such a case in 1941. He found only 340 cases up to 1939, distributed as follows:

Pancreatic tissue was found in the stomach in 31 per cent, duodenum in 31 per cent, jejunum in 21 per cent, ileum in 10 per cent. That found in the stomach was usually in the pyloric end and gave

symptoms simulating peptic ulcer, cholecystitis or pyloric obstruction.

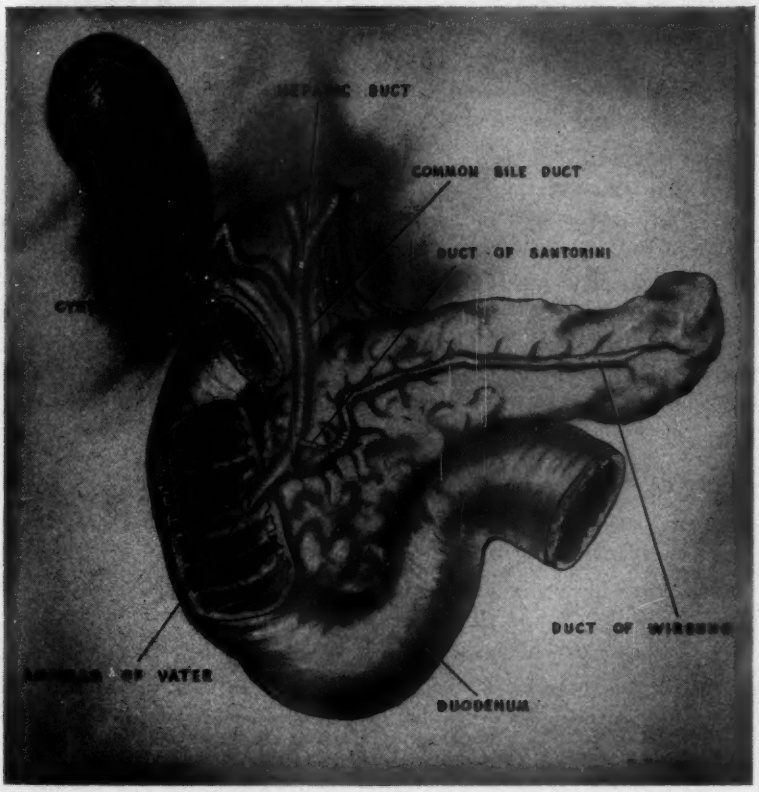

Fig. 1. Normal relationship of gallbladder, cystic, hepatic, common bile duct and pancreatic ducts.

The function of the pancreas is twofold—the manufacture of an internal secretion to assist in carbohydrate metabolism and the elaboration of an external secretion which contains important digestive ferments: namely, trypsinogen, diastase, renin and steapsin. The trypsinogen when poured into the duodenum is activated by the enterokinase into the very important ferment trypsin. Should this activation, for any reason, occur in the pancreas, it is considered a most likely cause of acute hemorrhagic pancreatitis. The internal secretion is known as insulin which comes from the Islets of Langerhans and is necessary in carbohydrate metabolism. Its absence or deficiency results in diabetes.

Trauma

The pancreas, situated deep in the abdomen as it is, rarely is injured by crushing blows to the abdomen. When such injury occurs, it is due to the pressure of the organ against the vertebral column as in car wheels passing over the body, crushing between two objects, etc. Stab wounds and gunshot wounds often result in hemorrhage and severe shock.

The symptoms of pancreatic injury are pain in the epigastrium, shock, nausea, vomiting and abdominal rigidity. In case of hemorrhage, dullness in the lateral wall of the abdomen appears after a sufficient time has allowed the blood to accumulate.

The treatment consists of prompt exploration of the abdomen, suture of the wound or control of hemorrhage by tamponade and drainage instituted. Fistulae often follow such procedures, but usually close spontaneously within 4 to 6 weeks. Reoperation is occasionally necessary to close the fistula.

Acute Hemorrhagic Pancreatitis

Actually, this condition is more of a necrosis than an infection. It is seen more often in men than in women and between the ages of 25 to 50. It is noted more in obese people.

Whatever may conduce to activate pancreatic enzymes within the pancreas will be an exciting factor in producing the acute pancreatitis. The concurrent destruction of blood vessels results in hemorrhage, with pressure necrosis and the pouring into the lesser peritoneal cavity of a chocolate-like fluid which in the early and intermediate stages of the disease is found to be sterile.

Reviewing briefly the anatomic structure of the common duct and the Duct of Wirsung, one can appreciate that obstruction at the Ampulla of Vater would cause a back flow of both bile and pancreatic juice which is 500 to 800 c.c. per day, into the Duct of Wirsung, as well as into the hepatic duct, and thus the activation of pancreatic enzymes is effected. The presence of bile in the pancreas results in activating trypsinogen into trypsin which is followed by pancreatic autodigestion and acute hemorrhagic pancreatitis.

Symptoms

Sudden onset of agonizing pain in the epigastrium, vomiting, prostration, and pain referred to the back, to the loins, and to the left scapular region. The pulse rate is rapid and steadily rises and the volume is weak. The blood pressure falls, and the skin is cold, clammy and cyanotic. Davis[5] stresses a clinical picture as corrobora-

tive of above findings in acute hemorrhagic pancreatitis—namely, a cyanotic color over the abdomen, a brownish discoloration posteriorly near the twelfth ribs on either side with a noticeable number of petechial patches on the buttocks as being almost pathognomonic. Death may occur within 36 hours. If the patient survives the acute attack, some of the symptoms subside but the secondary symptoms appear with high temperature, chills, leukocytosis and a tumor mass in the epigastrium more prominent on the left side.

Differential Diagnosis

Diagnosis should be made from appendicitis by localization of maximum tenderness after a short time in the right lower quadrant, from acute intestinal obstruction by slower onset and more pronounced vomiting of fecal matter after 24 to 36 hours, which does not occur in pancreatitis.

From acute cholecystitis, which pain is more pronounced to the right of the median line under the diaphragm and referred to the back and subscapular region. From perforated peptic ulcer by history of indigestion, rapid distention obliterating the liver dullness, and x-ray visualization of air under the diaphragm. In acute pancreatitis the urinary diastase rises rapidly to 250 units or more. The blood amylase concentration rises rapidly from a normal of 4 to 6 units.

A strong point in favor of acute pancreatitis is the marked increase in concentration of pancreatic enzymes in the blood. Both amylase and lipase tests are reliable and accurate. Comfort[6] found lipase concentration in the serum to be 99 per cent accurate and amylase to be 87 per cent accurate. Amylase must be performed early since necrosis of the pancreas will destroy the enzyme.

Urinary amylase determination is much less reliable. The finding of glycosuria and hyperglycemia in progressive readings indicates a grave prognosis. Serum calcium is decreased and when the concentration drops below 7 mg. per 100 c.c. of blood, the prognosis is fatal.

Treatment

Little time is usually afforded for scientific study of such a condition because most patients die rather promptly regardless of treatment. So soon as the initial shock recedes, exploratory laparotomy should be done.

The incision is supraumbilical in the midline. Thorough but quick inspection is made. If a blood tinged fluid with floating particles of fat necrosis is present, the pancreas should be felt and

usually a swollen enlargement is noted. Sometimes the lesser omental cavity is distended with either fluid or pus.

With this condition present and the patient in shock, the surgeon is prone to institute drainage by placing a penrose drain in each side of the cavity and a stab wound in the loin and close the abdomen. One should remember that such a temporary procedure does not remove the cause and, since obstruction at the sphincter of Oddi is responsible, or at least is present, in such a high percentage of acute pancreatitis, the gallbladder and common duct should be quickly, meticulously and thoroughly explored and, if a stone or other obstruction to the proper emptying of the bile and pancreatic secretion into the duodenum is found, such obstruction must be removed. If edema or other causes seem to be of prolonged duration, a cholecysto-gastrostomy or jejunostomy should be done, drainage instituted, and closure.

If acute pancreatitis without hemorrhage is differentiated from a slow leaking perforation of a peptic ulcer, or an attack of gallstone colic, operation should be performed, the lesser omentum separated and the peritoneum covering the pancreas should be incised transversely and widely. Drainage with rubber tissue must be adequate. If the gallbladder is edematous with jaundice or high icterus index present, it should be removed and the common duct explored for stones. A T tube should be left in the common duct.

If the head of the pancreas is hard and enlarged, with jaundice present, and the gallbladder is not too acute and edematous, then the bile should be diverted by uniting the gallbladder to the stomach or duodenum. Investigation and satisfactory exploration must still be made of the condition of the Ampulla of Vater for the probability of stone which by ball valve action is responsible for the back flow of bile and interferes with the proper drainage of the excretory duct of the pancreas.

Chronic Pancreatitis

Chronic inflammation of the pancreas becomes a surgical entity when diagnosis of disease in the biliary tract has been made, the removal of which might relieve the jaundice and permit the pancreas to regain its normal function.

Judd, of the Mayo Clinic, in 1921 reported a series of 1290 patients having gallbladder disease who also had an associated pancreatitis in 347 cases, or 26 per cent.

The fact must not be forgotten that arteriosclerosis, syphilis and tuberculosis play an important part in the etiology of this disease.

The symptoms are those of recurring attacks of indigestion, boring pain referred to the back or to the costal margins, nausea, occa-

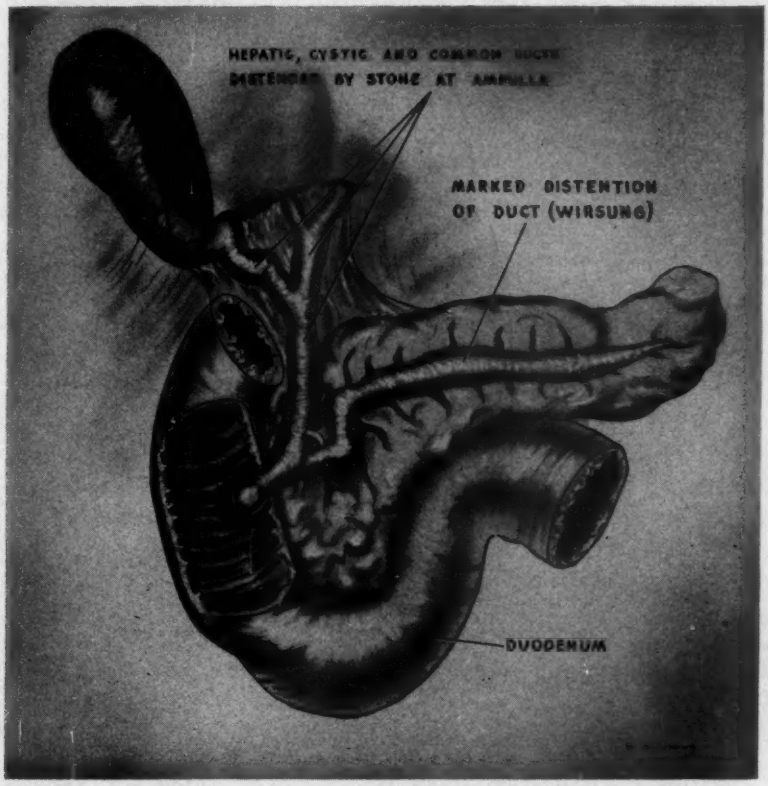

Fig. 2. Stone formation at the Ampulla of Vater, showing back flow of bile into the common, hepatic, cystic ducts and the Duct of Wirsung. Note marked distention with resulting pancreatitis.

sional vomiting, constipation, rarely visible jaundice in the early stages are all associated with the disease.

Treatment

Management of chronic pancreatitis consists in the treatment of the underlying cause. Medical care is important. Surgery directed to the biliary or gastric systems may remove the cause and, therefore, restore the pancreas to its normal function. Removal of the gallbladder here may be of questionable merit. If any suspicion of carcinoma of the pancreas exists, a cholecystogastrostomy should be performed.

Pancreatic Calculi

Francisco[7] reviewed the literature in 1942 and found only 250 cases of pancreatic calculi reported. These stones when found are composed largely of calcium carbonate and tribasic calcium phosphate.

Since normal pancreatic juice contains very little of these salts, it is thought that infection may alter the chemical composition of the juice and allow a deposition of calcium salts in the ducts.

They are usually seen in the head of the pancreas and may result in atrophy and sclerosis of the parenchyma of the organ, but the Islets remain free from involvement.

The calculi may be round, ovoid or irregular and vary greatly in size from grains of sand to 5 or 6 cm. in diameter. They are grayish white in color and are very brittle. They may also be deposited as sand formation throughout the parenchyma of the gland.

The symptoms are those of chronic pancreatitis. Since the stones are radio positive, x-ray is of marked value in diagnosis. Treatment is surgical removal and is very difficult.

Approach to the head of the pancreas is made by mobilizing the duodenum, entering the gastro-colic omentum, being exceedingly careful to preserve the middle colic artery. The tail, rarely involved, may be reached through the transverse meso-colon.

When the stone is identified by crepitus, careful blunt dissection and removal through the smallest opening possible is done. The cavity, if not infected, should be closed by sutures and the area drained for 48 hours.

Symptoms of Cysts

Retention cysts are most often found. They are not inordinately large and follow obstruction of the smaller pancreatic ducts from calculus or chronic pancreatitis. The fluid contains mucin cholesterol, fat and usually one or more pancreatic enzymes. They present a mass in the upper abdomen, either to the right or left, are seldom painful, may have pressure symptoms, dyspnea and jaundice.

Treatment

Regardless of the cause, the treatment of cysts of the pancreas is always surgical by marsupialization or extirpation. In the former, the incision is made over the most prominent part of the tumor, a small incision is made into the cyst wall and the fiuid evacuated.

The edges of the sac are sutured to the edges of the abdominal

Cysts of the Pancreas	Tumors of the Pancreas
Mayhorner's classification of the pancreatic cysts is as follows:	*Benign*
I. Cysts resulting from defective development	1. Adenoma
a. dermoid	2. Fibro-adenoma
b. inclusion	3. Lipoma
II. Traumatic cysts	4. Myxoma
III. Retention cysts	*Malignant*
IV. Neoplastic cysts	1. Carcinoma
a. Cystadenoma	2. Squamous cell carcinoma
b. Cystadenocarcinoma	3. Ductal adenocarcinoma
c. Teratomatous cyst	4. Acinar adenocarcinoma
V. Cysts resulting from parasites	5. Islet adenocarcinoma
	6. Sarcoma

wound and drainage instituted. These cyst fistulae may close spontaneously or may drain for many years. One of my own cases has been draining intermittently for 20 years, yet the patient has been apparently in good health otherwise during this time. Low sugar diet facilitates closure. In cystadenomas recurrence is the rule. Complete extirpation should always be done when possible.

These tumors are difficult and dangerous to remove. If a cyst wall is opened and the bottom of the cavity shows a papillomatous tumor, one may attempt extirpation but if the sac is smooth, one had better be content with marsupialization and drainage. This procedure is often followed by infection but this is the lesser of the two evils.

Carcinoma of the Pancreas

Carcinoma of the pancreas may be primary or secondary to extension from malignant disease in the stomach. It is usually found in the head and less frequently in the body and tail.

The symptoms are those of digestive disturbances, particularly starches and fats, loss of strength, weight and appetite and usually seen at the middle or later periods of life. The pains are deep, vague and radiate to the epigastrium and the back. Jaundice is a moderately late symptom unless the lesion is located near the common duct. It may, however, be the fist symptom which the patient regards seriously. Jaundice from pancreatic carcinoma is progressive, relentless, profound and persistent. Colicky-like seizures of epigastric distress are frequent in the early stages but he may be free for weeks or even longer, which deludes the patient into regard-

ing his transient attacks as mere indiscretion in diet. The rapid loss of weight in these cases soon permits the palpation of an epigastric tumor, non-conformatory to any patterns since more than the pancreas is usually involved. The tumor does not move with respiration. This is a sign of relatively great diagnostic significance.

X-ray is of little or no value. The perplexing problem to the surgeon is to determine whether the symptoms are from a chronic pancreatitis or carcinoma. They closely resemble each other in the early manifestations and surgical exploration is most usually required to differentiate the two conditions.

Watson's test has been of value in diagnosing the malignancy early enough for surgery to be helpful. It is as follows: In carcinoma of the pancreas, the fecal urobilinogen usually is *less* than 10 mg. per day. In obstruction from stone in the common duct it is usually 10 mg. or *more.*

In recent years the diagnosis has repeatedly been made early enough for radical surgical extirpation.

Treatment

If the diagnosis is made early enough, single stage removal is advised. If late or even mid-stage, the two stage procedure is preferable.

Removal of the body or tail of the pancreas is much more easily accomplished. In lesions of the head, much more consideration is required for the disposition of the bile and the pancreatic juice, both of which are partially or completely blocked by compression of the growth. If one stage is elected, the abdomen is opened, the survey made, and the common duct is ligated distal to the cystic duct. The gallbladder is then anastomosed to the stomach or jejunum. One then does a gastroenterostomy by the ante-colic method. The head of the pancreas and contiguous involved structures are removed in one block with the attached duodenum.

Hemostasis must be meticulous and complete. The open end of the pancreas and ducts are carefully closed with non-absorbable sutures and drainage instituted. This is a formidable procedure and only the best surgical risk should be selected for the operation. If it appears too much of a shock for the patient, a palliative cholecystoenterostomy should be done. This will take care of the flow of bile for a longer period of the disease than will cholecystogastrostomy in which event the bile would still have to pass the Ampulla of Vater which may be occluded along with the inflammatory process of the head of the pancreas. The mortality is high and

few patients live longer than 7 to 12 months after operation. X-ray therapy is of no value.

Report of Cases

White male, age 61, gave a history of prolonged distress in the upper abdomen with loss of weight, and the presence of a mass high in the abdomen. The blood picture was slightly lowered in relative ratio. The urine showed one plus sugar with no other positive findings. X-ray of the stomach showed position downward with a large ovoid shadow above the stomach.

Laparotomy was done. A large pancreatic cyst was found protruding through the gastro-hepatic omentum and, upon opening the abdomen, the cyst, including the stomach, literally forced itself into the wound on account of the tremendous pressure within the cyst. Careful packing around the cyst was done and aspiration by suction trocar, removed 2000 c.c. of a milky, somewhat viscid fluid.

Marsupilization was considered the procedure of choice and the edges of the cyst wall were securely fastened to the abdominal wall to prevent escape of fluid into the peritoneal cavity. The fluid did not excoriate the skin, showing little or no trypsin digestion of the skin.

The cyst was explored, which showed no diverticulæ but apparently a wide attachment along the body and tail of the pancreas. The inside of the cyst cavity was thoroughly painted with 10 per cent phenol in order to destroy the secreting membrane. Gauze pack was inserted and the wound closed. The patient made an uneventful recovery but a fistula persisted for a period of 3 years as seems to be the average in 15 per cent of the cases—antidiabetic diet was used. At the end of 3 years the wound was reopened, the cyst wall freed and, through the gastro-hepatic opening, a section of the body of the pancreas was removed, including the base of the sac. The wound in the pancreas was closed with nonabsorbable suture. The patient recovered promptly and it is now 2 years since the last operation and there is no evidence of fistula.

A second case, in a male, age 46, was operated on two and a half years ago. The secondary anemia was more pronounced, the patient was extremely weak and toxic. The same type of operative procedure was done but the contents proved to be a thick muco-purulent material. Examination showed no trypsin but low grade pyogenic organisms. Drainage was established and still persists after two and a half years. The gain in weight and strength has been satisfactory. Chemotherapy, transfusions and penicillin have all been used but without complete eradication of the infection.

Cultures are being made to determine the nature and virulency of the infection and, if satisfactory, a secondary operation will be performed.

It has been my privilege to remove tumors from the tail and distal part of the body of the pancreas, with successful results, all being of epithelial origin and benign. Removal of the head of the pancreas for carcinoma has been performed in two cases after the method described above, one patient living 2 months, the other 5 months after operation. The error in both the above cases was late diagnosis. With Watson's formula for early detection of malignancy of the pancreas, it is to be hoped that surgery may have more to offer this type of patient in the future. I repeat that the entire subject of pancreatic management needs much more study.

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CARCINOMA OF THE BREAST

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The treatment of carcinoma of the breast has progressed far since 1860 when Waldeyer first suggested that cancer spread through the lymphatics.[1] Perhaps the greatest milestone in the history of this disease was the work of Halsted. With his introduction of the radical surgical treatment, including the dissection of the axilla, in 1893 the results of treatment of malignancies of the breast greatly improved. Since then radical surgery has become the method most generally accepted by the medical profession.[2]

Nevertheless, advancements in our knowledge of this condition continue to be made. Sampson Handley advocated removal of more fascia and less skin, which proved clinically sound. The application of postoperative x-ray therapy has been found to increase the survival rate by Adair,[3] Harrington[4] and others.[9] Lately, the use of testosterone propionate has been advocated to prolong life and give more comfort to some of those who have this type of carcinoma.[5] Interestingly enough, castration of the male with carcinoma of the breast has been found to be of great benefit in far advanced cases.[8]

It has been estimated that no less than 50,000 women in the United States have cancer of the breast at the present time.[7] Adair[3] states that this type of cancer is increasing and Harrington[4] also finds the mortality rate is increasing. The breast is second only to the uterus as the most frequent site of carcinoma in women.

Needless to say, this disease still presents a challenge to the profession. While a cure rate of 60 per cent to 89 per cent can be obtained in cases without involvement of axillary nodes,[3] we find the mortality going up instead of down. Obviously the answer to this is early diagnosis and proper treatment. Educational programs such as those sponsored by The American Society for Control of Cancer are doing much to dispel superstition and bring the patients in for early examination. These programs should be encouraged. Then it becomes the obligation of the first examining physician to establish a definite diagnosis, or to see that a definite diagnosis is established, to be followed up by proper and adequate treatment.

Etiologic Factors

The ultimate answer to the etiology of cancer of the breast, like

many other forms of cancer, still remains somewhat of an enigma. Although the whole problem may not have been solved, certainly there are some pertinent facts that deserve thoughtful consideration. Geschickter et al have shown that all of the estrogens of sufficient potency for clinical use will produce mammary cancer in the rat regardless of chemical composition or physiologic potency.[10] Apropos to this, Auchincloss and Haagensen have reported a case of human cancer of the breast possibly induced by an estrogenic substance.[11] This in turn may have some relation to the enlightening discovery made by Kennaway and Cook in London that the carcinogenic substance dibenzanthracene is present in the estrogenic hormones.[8]

It is a well known fact that certain strains of mice have been developed that are highly susceptible to cancer of the breast and other strains that are highly resistant. Coincidental to this, Nathanson and Andervont found that the C_3H strain of mice which ordinarily develops cancer of the breast are prevented from doing so by the administration of testosterone at an early age.[8] Needless to say, the hereditary factor has been observed in humans by most physicians who see many cases of cancer of the breast.

The relation of benign lesions of the breast to cancer is an interesting one. Warren[12] found that in the age group from 30 to 49 years, patients with chronic mastitis and chronic cystic mastitis developed 11.7 times as much carcinoma of the breast as the Massachusetts female population of comparable age. Clagett et al found similar results.[16] Geschickter[13] found that 3 per cent of 201 patients who had had excision of a fibroadenoma subsequently developed a malignant tumor of the breast.

Diagnosis

The utmost significance must be given to the painless lump in the breast that does not go away. It has been reported that 90 per cent of such cases represent cancer if the patient is past 40 years of age.[7] The lump has added significance if it is in the upper outer quadrant of the breast. Attachment of the skin is characteristic, but not pathognomonic because certain benign lesions may cause some degree of this. Nevertheless 75 to 80 per cent of malignant breasts have this feature. This again is not an early sign because 72.6 per cent of such cases have metastases to the regional lymph nodes.[4]

Ulceration of the nipple may be a simple eczema, a mild dermatitis or Paget's disease. Paget's disease is an intraductal carcinoma of the upper ducts, which will metastasize. This condition presents

findings typically described by Cheatle, Ewing and others as a chronic red scaly or granular superficial ulceration about one nipple. These cases are candidates for radical surgery.

Some authors report that malignancy is back of 50 per cent of bleeding from the nipple.[7] If the lesion is not malignant, it may well be a papilloma of one of the ducts. These are premalignant and should be excised, care being taken to have the benefit of immediate pathologic examination, so that if malignancy is reported, a radical mastectomy may be done.

Pain occurs only in 8 to 10 per cent of cancer of the breast as a presenting symptom.[4] Most commonly, women have delayed coming to the doctor because there was no pain. Pain may and usually does mean cystic mastitis. It is not the usual early symptom of cancer.

A careful examination should be made. The breasts are inspected with the patient sitting up. Even an early cancer may cause distortion of the breast outline. The slightest dimpling or furrowing of the skin over the lump is significant. Palpation should be made while the patient is sitting and again in the recumbent position, the gland pressed against the chest wall, but not grasped between the thumb and fingers. Next the axilla should be carefully examined with the arm relaxed in different positions, particular emphasis given to the thoracic surface, because the earliest nodes are usually felt there. Transillumination may be of help in the differentiation of cysts of the breast. Before treatment is started an x-ray of the chest should be made routinely because even the smaller cancers sometimes show x-ray evidence of metastases.

Treatment

Hermann[14] states that about 11 per cent of the cases seen at the Memorial Hospital in New York are inoperable. Patients with any of the following conditions are considered inoperable and are treated by palliative x-ray therapy and not by surgery, that is after the lesion is proven by histologic examination.

1. Extensive edema of the skin of the breast.
2. Metastatic nodules in the skin of the breast and chest wall.
3. Edema of the arm.
4. Firm fixation of the tumor mass to the chest wall.
5. Supraclavicular or cervical metastases.
6. Inflammatory type of carcinoma.
7. Distant metastases.

In some cases where the tumor seems fixed to the chest wall and there are no demonstrable metastases, x-ray therapy may be given and then the case reconsidered for possible surgery later. However, it was found that routine preoperative irradiation did not increase the percentage of 5 year survivals in the cases studied at the Memorial Hospital.[4] It has been given up by most men. On the other hand, a definite increase in the 5 year survivals has been reported by postoperative irradiation in those who have axillary metastases. This is recommended routinely.[4,14,15,17]

Having made a diagnosis of an operable carcinoma of the breast, the treatment of choice is radical mastectomy. The value of two separate operating set-ups is stressed, one for the local excision or biopsy of the tumor for immediate frozen section examination and the other for the radical mastectomy. While the biopsy wound is being closed tightly, the pathologic report is received. If cancer is reported, the breast is prepared again and the operative field redraped with fresh linen. Different gowns, gloves and instruments are used for the second procedure. The importance of this step has recently been emphasized by Brandes et al,[18] who reported a case of accidental transplantation of cancer in the operating room to the donor site of a skin graft, by failing to change gloves following the biopsy.

After the operative field has been freshly prepared, the skin incision is outlined. There are many types of skin incisions that have been advocated. We prefer the Willy Meyer type incision which gives all of the exposure needed and gives a good functional result. This extends from the shoulder anteriorly around the mass in the breast and down over the upper rectus muscle.

The skin flaps are dissected from the underlying subcutaneous tissue, leaving only enough tissue beneath the skin to maintain adequate blood supply. The dissection of the flaps is carried out around the entire operative field, the medial flap dissected just past the midline and the lateral flap carried to the border of the latissimus dorsi muscle. The attachment of the sternal portion of the pectoralis major muscle is severed from its attachment to the humerus. The lymph nodes along the upper border of the brachial vessels are thoroughly removed and the dissection is carried to the lower border of the pectoralis minor muscle which is cut loose from its insertion to the coracoid process of the scapula in preparation for its removal.

The axillary, subclavicular and subscapular nodes are removed in one mass including all of the lymph nodes and lymph bearing fascia both above and beneath the axillary vessels and nerves. All

of the nodes in the infraclavicular fossa are carefully removed at the point where the axillary vein enters the thorax.

The long thoracic nerve to the serratus muscle as well as the middle subscapular nerve to the latissimus dorsi are preserved when this is possible. An attempt is made also to preserve the subscapular artery and vein accompanying the nerve. This is done to preserve the collateral circulation, thus preventing swelling of the arm.

As these structures, including the breast, subcutaneous tissue, axillary, subscapular and subclavicular nodes as well as the node bearing fascia, pectoralis minor and major muscles, with accompanying sheaths, lymph vessels and nodes are swept downward and lateralward off of the chest wall, the vessels and nerves of the axilla are kept covered with warm wet saline gauze packs. The origins of the pectoralis muscles are cleanly dissected from the thorax, as well as the sheaths of serratus muscle. The dissection continues with the removal of the upper anterior rectus muscle sheath and all of the structures are removed together in one specimen. Careful hemostasis is maintained, tying the perforating branches of the internal mammary and intercostal vessels close to the chest wall.

The wound is closed with interrupted silk with a drain placed in the lower end of the wound and one through a stab wound in the axilla. There should be slight tension in the skin to close dead space. The wound is dressed and well padded in the axilla with pressure and the arm is immobilized for 2 or 3 days in a Velpeau bandage. The initial dressing is done in about 5 days at which time the drains are removed.

Lachman has pointed out the paths of metastatic spread of cancer of the breast, giving a sound background for the extensive operation outlined above.[19]

As soon as the wound has healed solidly the patient is referred for postoperative x-ray therapy. X-ray castration is also recommended for those who had a high grade of malignancy or involvement of the axilla, even in some women who have ceased to menstruate. This has been proved to be of definite value by Adair et al.[20] Horsley has suggested bilateral oophorectomy,[21] but this would seem to involve more risk and hardly necessary. On the other hand surgical castration of the male has been found to be of benefit in far advanced cancer of the breast.[22]

Prudente has advocated postoperative administration of testosterone, finding more survivals and less recurrence in his series of

female patients.[6] It will be interesting to see the role testosterone will eventually play in this disease. Certainly it has proven its value in some cases of far advanced disease with metastases, as reported by Adair and Hermann.[5] My own experience with its administration in carcinoma of the breast with metastases has been very encouraging. However, it has been my policy to use it in conjunction with x-ray therapy.

There are certain factors which influence the eventual prognosis of this disease in any stage of growth. Haagensen has found that if the carcinoma develops during pregnancy or lactation, the chances of cure are quite poor. He reports that 95 per cent have axillary metastases and there are only 15 per cent 5 year survivals.[23] Contrary to the accepted idea, de Cholnoky finds very little difference in the survival rate in women under 30, as opposed to the older age group.[24] However, most investigators feel that the prognosis is considerably worse in the younger women.

The following is a table compiled from the literature[3-25] to show a comparison of 5 year results of different authors, some using surgery alone and others using surgery and postoperative irradiation.

An analysis of this table reveals little difference between the cases treated by surgery alone and those having surgery and postoperative irradiation when the lesion has not metastasized to the axilla. However, there seems to be a definite increase in the 5 year results with the combination of surgery and postoperative irradiation when axillary metastases are found.

The use of x-ray therapy in no way lessens the necessity for meticulous handling of tissues, or allows for removal of smaller amounts of tissue, or the leaving of any of the axillary contents, as is advocated in some quarters today. If surgery is chosen, one is obligated to do as complete an operation as possible.

We are heartily in agreement with Adair who states that "the advocates of local mastectomy to be followed by irradiation, for breast cancer, have done great harm because they have opened wide the gates for inadequate surgical procedures for the cure of a lethal disease, and have confused many."[3]

Summary

Early diagnosis followed by adequate treatment offers a good chance for cure in patients with carcinoma of the breast. However,

AUTHOR	Axillary Nodes Not Involved		Axillary Nodes Involved	
	Operation Without Irradiation	Operation With Irradiation	Operation Without Irradiation	Operation With Irradiation
Greenough & Simmons	56 Per Cent		24 Per Cent	
Sistrunk & McCarthy	65 Per Cent		22 Per Cent	
Peck & White	65 Per Cent		23 Per Cent	
Mathews	64 Per Cent		29 Per Cent	
Bloodgood	70 Per Cent		20 Per Cent	
White	70 Per Cent		19 Per Cent	
Westermark		60 Per Cent		46.6 Per Cent
Schreiner		63 Per Cent		23 Per Cent
Schmitz		64 Per Cent		42 Per Cent
Harrington		66.2 Per Cent		22.3 Per Cent
Adair		72 Per Cent		23 Per Cent
Pfahler & Parry				47 Per Cent
Eggers				33.3 Per Cent

this lethal disease must be treated radically to get a high percentage of cures. Radical mastectomy with clean dissection of the axilla, resection of the pectoralis muscles and the lymph bearing fascias of the chest wall has been the treatment of choice since the time of Halsted. A further increase in the survival rate has been obtained by postoperative irradiation with the aid of x-ray castration in some cases. The administration of testosterone propionate has been proven to be of some aid in the treatment of far advanced cases. The use of hormones in the treatment of this disease will bear more investigation. It will be interesting to see their eventual role in the treatment of carcinoma of the breast as well as in other carcinomas that have a close relationship to the internal secretions.

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THE PARATHYROID PROBLEM

Due to revised criteria for diagnosis, parathyroid adenoma has been found to be more common than formerly believed. The older concept, which stubbornly persists, that, in order to diagnose parathyroidism, one must have typical findings of osteitis fibrosa cystica, should be discarded. The most recent work indicates that bone disease manifestation is not only the least common clinical finding but is often far advanced and of long duration before the patient is finally properly diagnosed.

Albright, in 1934, reported 17 proved cases seen at the Massachusetts General Hospital over a period of 2 years. And, in 1942, Cope extended the report at the same institution to cover 67 cases seen in 10 years. Others, guided by these startling revelations, have become parathyroid "conscious" and are proving the presence of this condition more frequently than heretofore supposed. Thus, Cook at the Mayo Clinic, reported 24 proved cases in a 2½ year period. Subsequent to this time covering the years 1945 and 1946, an additional 24 cases have been seen. At the same institution, during the preceding 13 years, only 14 cases had been diagnosed. I therefore believe that the attention of the profession should be arrested to this incapacitating and ofttimes serious malady, which can be both easily diagnosed and dramatically remedied by surgery.

SYMPTOMATOLOGY

It is well to have organized thoughts to serve as clinical guidance in considering the symptoms of the disease. Renal calculi exist in

about 90 per cent, bone changes occur in about 60 per cent, of proved parathyroid adenoma. Of those cases with bone changes, about 50 per cent have minimal findings of osteoporosis and the remainder show more extensive decalcification in varying amounts. The formation of renal calculi, nephrocalcinosis and osseus disease are the body's expression of negative calcium balance. The symptoms derive from this chemical abnormality. As one would expect, they are opposite to those seen in tetany. Fatigue, peplessness, lack of stamina, anorexia, polyuria, and constipation are common complaints. Renal colic occurs during stone passage and forcibly calls attention to the possibility of the presence of parathyroidism. Vague pains of skeletal origin and pathologic fractures are only present when there is extensive osteoporosis and occur fairly late.

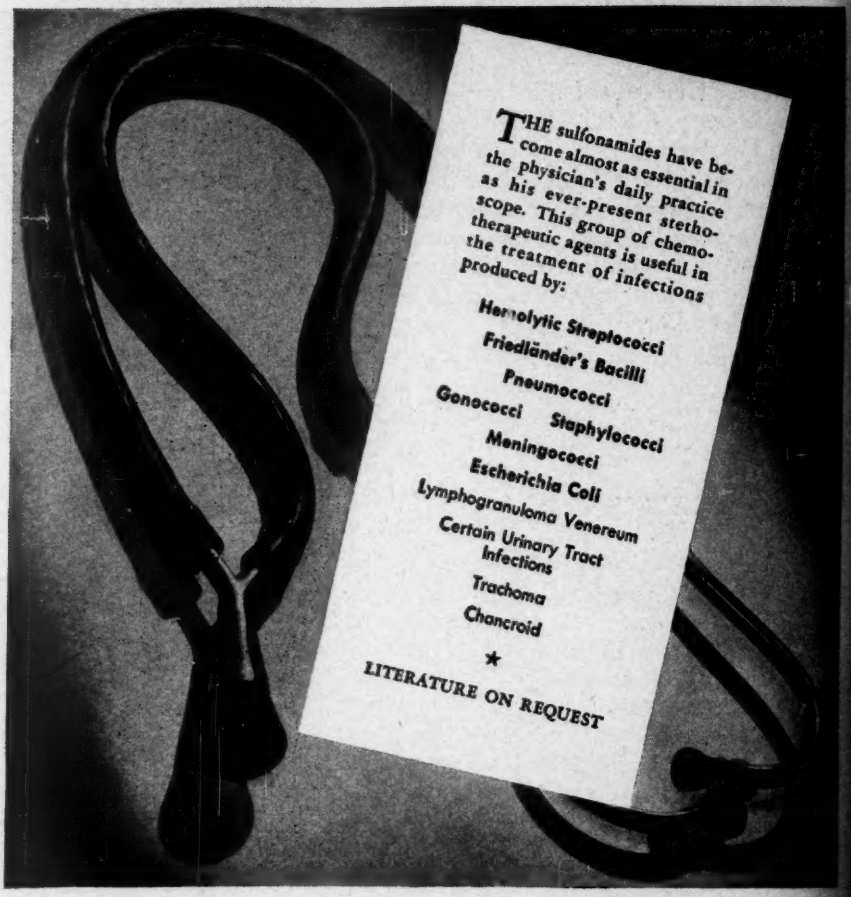
Diagnosis

When once suspected the diagnosis is made almost with certainty by laboratory methods. Since the primary pathology concerns depletion of the skeletal calcium, there exists a smaller calcium intake than urinary output (negative calcium balance), an elevated serum calcium and lowered phosphorus. Accordingly, if on 3 successive days while on a weighed calcium intake (125 mg. per day) there is a 24 hour urinary output of 125 to 200 mg., a serum calcium which averages from 11 mg. per cent and upwards (frequent determinations are often necessary) and a lowered inorganic phosphorus (less than 3 mg. per cent, the diagnosis is established. Confirmative evidence may sometimes be obtained by roentgenograms. The mandible is one of the earliest bones to reveal demineralization and is also advantageous to study as the teeth do not decalcify and therefore the contrast is striking. There is loss of the trabecula arrangement, a diffuse hazy demineralization and disappearance of the lamina dura. The x-ray findings may proceed to the extensive changes seen in well advanced and typical cases of osteitis fibrosa cystica.

The urologic features of the disease are not pathognomonic insofar as diagnosis is concerned. Not all individuals with calculous disease have concomitant parathyroidism. However, the presence of urinary calcification is often manifest by an explosive outburst of pain and is the cause for seeking medical aid. Since calculous disease is present in 90 per cent of parathyroid adenoma it would prove profitable to study all these cases from this point of view. When it is realized that 30 per cent of patients with calculous disease ultimately have their kidneys sacrificed and that the kidneys are vital organs, then appraisal of the parathyroid glands in all cases of urinary calculi can be appreciated.

The first cases of this disease were described and diagnosed in this country in 1929. And, not unlike many other momentous medical discoveries, the true importance and detailed evaluation has taken nearly 15 years. It is the purpose of this article to alert surgeons to the relative frequency of a condition which has been and still is regarded by many as a rarity. Only by bearing in mind the possibility of parathyroid adenoma and by possessing the curiosity to pursue the laboratory studies, will the diagnosis be made as often as it should be.

CHARLES RIESER, M. D.



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